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PASSWORD:
TERMINAL (ENTER 1, 2, 3, OR ?):2


* * * * * * * * * *   Welcome to STN International   * * * * * * * * * *

 NEWS  1           Web Page URLs for STN Seminar Schedule - N. America
 NEWS  2           "Ask CAS" for self-help around the clock
 NEWS  3  SEP 09   CA/CAplus records now contain indexing from 1907 to the
                   present
 NEWS  4  AUG 05   New pricing for EUROPATFULL and PCTFULL effective
                   August 1, 2003
 NEWS  5  AUG 13   Field Availability (/FA) field enhanced in BEILSTEIN
 NEWS  6  AUG 18   Data available for download as a PDF in RDISCLOSURE
 NEWS  7  AUG 18   Simultaneous left and right truncation added to PASCAL
 NEWS  8  AUG 18   FROSTI and KOSMET enhanced with Simultaneous Left and Righ
                   Truncation
 NEWS  9  AUG 18   Simultaneous left and right truncation added to ANABSTR
 NEWS 10  SEP 22   DIPPR file reloaded
 NEWS 11  DEC 08   INPADOC: Legal Status data reloaded
 NEWS 12  SEP 29   DISSABS now available on STN
 NEWS 13  OCT 10   PCTFULL: Two new display fields added
 NEWS 14  OCT 21   BIOSIS file reloaded and enhanced
 NEWS 15  OCT 28   BIOSIS file segment of TOXCENTER reloaded and enhanced
 NEWS 16  NOV 24   MSDS-CCOHS file reloaded
 NEWS 17  DEC 08   CABA reloaded with left truncation
 NEWS 18  DEC 08   IMS file names changed
 NEWS 19  DEC 09   Experimental property data collected by CAS now available
                   in REGISTRY
 NEWS 20  DEC 09   STN Entry Date available for display in REGISTRY and CA/CAplus

 NEWS EXPRESS  NOVEMBER 14 CURRENT WINDOWS VERSION IS V6.01c, CURRENT
               MACINTOSH VERSION IS V6.0b(ENG) AND V6.0Jb(JP),
               AND CURRENT DISCOVER FILE IS DATED 23 SEPTEMBER 2003
 NEWS HOURS    STN Operating Hours Plus Help Desk Availability
 NEWS INTER    General Internet Information
 NEWS LOGIN    Welcome Banner and News Items
 NEWS PHONE    Direct Dial and Telecommunication Network Access to STN
 NEWS WWW      CAS World Wide Web Site (general information)

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FILE 'HOME' ENTERED AT 17:10:14 ON 10 DEC 2003

=> b medline caplus lifesci embase uspatfull biosis
COST IN U.S. DOLLARS                                SINCE FILE      TOTAL
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                                                        ENTRY    SESSION
FULL ESTIMATED COST                                      0.21       0.21

FILE 'MEDLINE' ENTERED AT 17:10:36 ON 10 DEC 2003

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FILE 'BIOSIS' ENTERED AT 17:10:36 ON 10 DEC 2003
COPYRIGHT (C) 2003 BIOLOGICAL ABSTRACTS INC.(R)

=> s snorna
L1          1549 SNORNA

=> l1 and tar
L1 IS NOT A RECOGNIZED COMMAND
The previous command name entered was not recognized by the system.
For a list of commands available to you in the current file, enter
"HELP COMMANDS" at an arrow prompt (=>).

=> s l1 and tar
L2             4 L1 AND TAR

=> dup rem l2
PROCESSING COMPLETED FOR L2
L3             2 DUP REM L2 (2 DUPLICATES REMOVED)

=> d l3 ibib abs tot

L3   ANSWER 1 OF 2     MEDLINE on STN                    DUPLICATE 1
ACCESSION NUMBER:    2003372791      IN-PROCESS
DOCUMENT NUMBER:     22789097   PubMed ID: 12907142
TITLE:               Inhibition of HIV-1 infection by lentiviral vectors
                     expressing Pol III-promoted anti-HIV RNAs.
AUTHOR:              Li Ming-Jie; Bauer Gerhard; Michienzi Alessandro; Yee
                     Jiing-Kuan; Lee Nan-Sook; Kim James; Li Shirley; Castanotto
                     Daniela; Zaia John; Rossi John J
CORPORATE SOURCE:    Division of Molecular Biology, Beckman Research Institute
                     of the City of Hope, Duarte, California 91010, USA.
CONTRACT NUMBER:     AI29329 (NIAID)
     AI42552 (NIAID)
     AI46030 (NIAID)
SOURCE:              MOLECULAR THERAPY, (2003 Aug) 8 (2) 196-206.
                     Journal code: 100890581. ISSN: 1525-0016.
PUB. COUNTRY:        United States
DOCUMENT TYPE:       Journal; Article; (JOURNAL ARTICLE)
LANGUAGE:            English
FILE SEGMENT:        IN-PROCESS; NONINDEXED; Priority Journals
ENTRY DATE:          Entered STN: 20030809
                     Last Updated on STN: 20030819
AB   A primary advantage of lentiviral vectors is their ability to pass through
     the nuclear envelope into the cell nucleus thereby allowing transduction
     of nondividing cells.  Using HIV-based lentiviral vectors, we delivered an
```

anti-CCR5 ribozyme (CCR5RZ), a nucleolar localizing **TAR** RNA decoy, or Pol III-expressed siRNA genes into cultured and primary cells. The CCR5RZ is driven by the adenoviral VA1 Pol III promoter, while the human U6 snRNA Pol III-transcribed **TAR** decoy is embedded in a U16 **snoRNA** (designated U16TAR), and the siRNAs were expressed from the human U6 Pol III promoter. The transduction efficiencies of these vectors ranged from 96-98% in 293 cells to 15-20% in primary PBMCs. A combination of the CCR5RZ and U16TAR decoy in a single vector backbone gave enhanced protection against HIV-1 challenge in a selective survival assay in both primary T cells and CD34(+)-derived monocytes. The lentiviral vector backbone-expressed siRNAs also showed potent inhibition of p24 expression in PBMCs challenged with HIV-1. Overall our results demonstrate that the lentiviral-based vectors can efficiently deliver single constructs as well as combinations of Pol III therapeutic expression units into primary hematopoietic cells for anti-HIV gene therapy and hold promise for stem or T-cell-based gene therapy for HIV-1 infection.

L3 ANSWER 2 OF 2 USPATFULL on STN

ACCESSION NUMBER:	1999:81758 USPATFULL		
TITLE:	Non-activated receptor complex proteins and uses thereof		
INVENTOR(S):	Davis, Roger J., Princeton, MA, United States Galcheva-Gargova, Zoya, Worcester, MA, United States		
PATENT ASSIGNEE(S):	University of Massachusetts, Boston, MA, United States (U.S. corporation)		
	NUMBER	KIND	DATE
PATENT INFORMATION:	US 5925566		19990720
APPLICATION INFO.:	US 1997-870518		19970606 (8)
	NUMBER		DATE
PRIORITY INFORMATION:	US 1996-19219P		19960606 (60)
DOCUMENT TYPE:	Utility		
FILE SEGMENT:	Granted		
PRIMARY EXAMINER:	Campell, Bruce R.		
ASSISTANT EXAMINER:	Nguyen, Dave Trong		
LEGAL REPRESENTATIVE:	Fish & Richardson, P.C.		
NUMBER OF CLAIMS:	23		
EXEMPLARY CLAIM:	1		
NUMBER OF DRAWINGS:	22 Drawing Figure(s); 18 Drawing Page(s)		
LINE COUNT:	2438		

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

AB The invention features a substantially pure ZPR1 polypeptide. For example, a ZPR1 polypeptide that specifically binds to a non-activated membrane-bound receptor (e.g., EGF or PDGF receptors) and specifically binds small nucleolar RNAs (e.g., U3). ZPR1 polypeptides can be isolated from any eukaryote, including mammals (e.g. rodents and humans) and fungi (e.g., S. cerevisiae and S. pombe).

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

=> d kwic tot

L3 ANSWER 1 OF 2 MEDLINE on STN DUPLICATE 1

AB . . . nucleus thereby allowing transduction of nondividing cells. Using HIV-based lentiviral vectors, we delivered an anti-CCR5 ribozyme (CCR5RZ), a nucleolar localizing **TAR** RNA decoy, or Pol III-expressed siRNA genes into cultured and primary cells. The CCR5RZ is driven by the adenoviral VA1 Pol III promoter, while the human U6 snRNA Pol III-transcribed **TAR** decoy is embedded in a U16 **snoRNA** (designated U16TAR), and the siRNAs were expressed from the

human U6 Pol III promoter. The transduction efficiencies of these vectors. . .

L3 ANSWER 2 OF 2 USPATFULL on STN
SUMM "Small nucleolar RNA" or "**snoRNA**" refers to RNA sequences located in the nucleolus and includes the sequences encoded by the U3, U10, U15, U22, and. . .
DETD . . . U10, U15, U22, U33, or the viral RNAs corresponding to the RRE (HIV Rev responsive element), HIV trans-acting response element (**TAR**), and adenovirus VA-RNA1 (Akusjavari et al., Proc. Natl. Acad. Sci. USA 77:2424-2428 (1980); Feng and Holland, Nature 334:165-167 (1988); Bartel. . . was investigated by this method. No binding of ZPR1 was detected in experiments using viral RNA corresponding to RRE, the **TAR**, or adenovirus VA-RNA1. In contrast, binding to ZPR1 was detected in experiments using several small nucleolar RNAs (snoRNAs), including U3,. . .
DETD The specificity of the interaction of RNA with ZPR1 was examined in greater detail in competition experiments using the **snoRNA**, U3. A [.sup.32 P]-labeled U3 probe was incubated with ZPR1 alone or in the presence of excess non-radioactive U3, anti-sense. . .

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=> d history

     (FILE 'HOME' ENTERED AT 17:10:14 ON 10 DEC 2003)

     FILE 'MEDLINE, CAPLUS, LIFESCI, EMBASE, USPATFULL, BIOSIS' ENTERED AT
     17:10:36 ON 10 DEC 2003
L1          1549 S SNORNA
L2             4 S L1 AND TAR
L3             2 DUP REM L2 (2 DUPLICATES REMOVED)

=> s l1 and py<2001
   3 FILES SEARCHED...
L4          991 L1 AND PY<2001

=> s l4 and tat
L5            2 L4 AND TAT

=> dup rem l5
PROCESSING COMPLETED FOR L5
L6            2 DUP REM L5 (0 DUPLICATES REMOVED)

=> d l6 ibib abs tot

L6   ANSWER 1 OF 2  USPATFULL on STN
ACCESSION NUMBER:        1999:81758  USPATFULL
TITLE:                   Non-activated receptor complex proteins and uses
                         thereof
INVENTOR(S):             Davis, Roger J., Princeton, MA, United States
                         Galcheva-Gargova, Zoya, Worcester, MA, United States
PATENT ASSIGNEE(S):      University of Massachusetts, Boston, MA, United States
                         (U.S. corporation)

                              NUMBER        KIND    DATE
                         ---------------- ------ --------
PATENT INFORMATION:      US 5925566                19990720            <--
APPLICATION INFO.:       US 1997-870518            19970606  (8)

                               NUMBER         DATE
                         ------------------ --------
PRIORITY INFORMATION:    US 1996-19219P     19960606 (60)
DOCUMENT TYPE:           Utility
FILE SEGMENT:            Granted
PRIMARY EXAMINER:        Campell, Bruce R.
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ASSISTANT EXAMINER: Nguyen, Dave Trong
LEGAL REPRESENTATIVE: Fish & Richardson, P.C.
NUMBER OF CLAIMS: 23
EXEMPLARY CLAIM: 1
NUMBER OF DRAWINGS: 22 Drawing Figure(s); 18 Drawing Page(s)
LINE COUNT: 2438
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB The invention features a substantially pure ZPR1 polypeptide. For example, a ZPR1 polypeptide that specifically binds to a non-activated membrane-bound receptor (e.g., EGF or PDGF receptors) and specifically binds small nucleolar RNAs (e.g., U3). ZPR1 polypeptides can be isolated from any eukaryote, including mammals (e.g. rodents and humans) and fungi (e.g., S. cerevisiae and S. pombe).

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L6 ANSWER 2 OF 2 MEDLINE on STN
ACCESSION NUMBER: 1999449717 MEDLINE
DOCUMENT NUMBER: 99449717 PubMed ID: 10518602
TITLE: Rev-mediated nuclear export of RNA is dominant over nuclear retention and is coupled to the Ran-GTPase cycle.
AUTHOR: Fischer U; Pollard V W; Luhrmann R; Teufel M; Michael M W; Dreyfuss G; Malim M H
CORPORATE SOURCE: Institut fur Molekularbiologie und Tumorforschung, Marburg, Germany.. ufischer@biochem.mpg.de
CONTRACT NUMBER: GM17699 (NIGMS)
SOURCE: NUCLEIC ACIDS RESEARCH, **(1999 Nov 1)** 27 (21) 4128-34.
Journal code: 0411011. ISSN: 1362-4962.
PUB. COUNTRY: ENGLAND: United Kingdom
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals; AIDS
ENTRY MONTH: 199912
ENTRY DATE: Entered STN: 20000113
Last Updated on STN: 20021218
Entered Medline: 19991202
AB The human immunodeficiency virus type-1 Rev protein induces the nuclear export of intron-containing viral mRNAs that harbor its binding site, the Rev response element (RRE). A leucine-rich region of Rev, the activation domain, is essential for function and has been shown to be a nuclear export signal (NES). Although Rev exports viral RNAs that resemble cellular mRNAs, competition studies performed using microinjected Xenopus laevis oocytes have previously indicated that Rev utilizes a non-mRNA export pathway. Here, we show that Rev is able to induce the export of both spliceable and non-spliceable RRE-containing pre-mRNAs and that this activity is not dependent on the location of the RRE within the RNA. Importantly, even RNA molecules of different classes, such as U3 **snoRNA** and U6 snRNA, which are retained in the nucleus by non-pre-mRNA mechanisms, are exported to the cytoplasm in response to Rev. Consistent with the notion that Rev-mediated export of RRE-containing RNA is mechanistically distinct from the export of processed cellular mRNA, a chimeric Rev protein in which its NES is replaced by the NES of hnRNP A1 does not induce the export of a Rev-responsive mRNA. Finally, we demonstrate that Rev/RRE-activated RNA export is, like other nuclear export pathways, linked to the Ran-GTPase cycle.

=> d l6 ibib kwic tot

L6 ANSWER 1 OF 2 USPATFULL on STN
ACCESSION NUMBER: 1999:81758 USPATFULL
TITLE: Non-activated receptor complex proteins and uses thereof
INVENTOR(S): Davis, Roger J., Princeton, MA, United States

	Galcheva-Gargova, Zoya, Worcester, MA, United States
PATENT ASSIGNEE(S):	University of Massachusetts, Boston, MA, United States (U.S. corporation)

	NUMBER	KIND	DATE	
PATENT INFORMATION:	US 5925566		19990720	<--
APPLICATION INFO.:	US 1997-870518		19970606 (8)	

	NUMBER	DATE
PRIORITY INFORMATION:	US 1996-19219P	19960606 (60)

DOCUMENT TYPE:	Utility
FILE SEGMENT:	Granted
PRIMARY EXAMINER:	Campell, Bruce R.
ASSISTANT EXAMINER:	Nguyen, Dave Trong
LEGAL REPRESENTATIVE:	Fish & Richardson, P.C.
NUMBER OF CLAIMS:	23
EXEMPLARY CLAIM:	1
NUMBER OF DRAWINGS:	22 Drawing Figure(s); 18 Drawing Page(s)
LINE COUNT:	2438

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

PI US 5925566 19990720 <--

SUMM "Small nucleolar RNA" or "**snoRNA**" refers to RNA sequences located in the nucleolus and includes the sequences encoded by the U3, U10, U15, U22, and. . .

DETD The specificity of the interaction of RNA with ZPR1 was examined in greater detail in competition experiments using the **snoRNA**, U3. A [.sup.32 P]-labeled U3 probe was incubated with ZPR1 alone or in the presence of excess non-radioactive U3, anti-sense. . .

DETD . . . 1312AG GCC CAC
#Phe Ile Met Asn Asp Pro Met Lys Ala His
405
#GCA CCT GAA GAC GAT CCA 1360AT GTG **TAT**
#Ala Pro Glu Asp Asp Pro Gln Asn Val Tyr
420
#TTT GAC CAA AAT GAG GAG 1408AA CGC ACC
#Phe Asp Gln Asn Glu Glu Tyr Lys Arg Thr
#440
#**TAT** GAG GCG GGC CTG GCC 1456CA GAG GGC
#Tyr Glu Ala Gly Leu Ala Lys Thr Glu Gly
455
- CCA CAG. . .

L6 ANSWER 2 OF 2 MEDLINE on STN

ACCESSION NUMBER:	1999449717 MEDLINE
DOCUMENT NUMBER:	99449717 PubMed ID: 10518602
TITLE:	Rev-mediated nuclear export of RNA is dominant over nuclear retention and is coupled to the Ran-GTPase cycle.
AUTHOR:	Fischer U; Pollard V W; Luhrmann R; Teufel M; Michael M W; Dreyfuss G; Malim M H
CORPORATE SOURCE:	Institut fur Molekularbiologie und Tumorforschung, Marburg, Germany.. ufischer@biochem.mpg.de
CONTRACT NUMBER:	GM17699 (NIGMS)
SOURCE:	NUCLEIC ACIDS RESEARCH, **(1999 Nov 1)** 27 (21) 4128-34. Journal code: 0411011. ISSN: 1362-4962.
PUB. COUNTRY:	ENGLAND: United Kingdom
DOCUMENT TYPE:	Journal; Article; (JOURNAL ARTICLE)
LANGUAGE:	English
FILE SEGMENT:	Priority Journals; AIDS
ENTRY MONTH:	199912
ENTRY DATE:	Entered STN: 20000113 Last Updated on STN: 20021218 Entered Medline: 19991202

SO NUCLEIC ACIDS RESEARCH, **(1999 Nov 1)** 27 (21) 4128-34.
Journal code: 0411011. ISSN: 1362-4962.
AB . . . dependent on the location of the RRE within the RNA. Importantly, even RNA molecules of different classes, such as U3 **snoRNA** and U6 snRNA, which are retained in the nucleus by non-pre-mRNA mechanisms, are exported to the cytoplasm in response to. .
.
CT . . .
GE, genetics
Exons: GE, genetics
Gene Products, rev: CH, chemistry
Gene Products, rev: GE, genetics
*Gene Products, rev: ME, metabolism
Gene Products, tat: GE, genetics
HIV-1: GE, genetics
Heterogeneous-Nuclear Ribonucleoproteins
Introns: GE, genetics
Mutation
Oocytes: ME, metabolism
RNA: GE, genetics
. . .
CN 0 (Gene Products, rev); 0 (Gene Products, **tat**); 0 (Heterogeneous-Nuclear Ribonucleoproteins); 0 (RNA Precursors); 0 (RNA, Small Nuclear); 0 (RNA, Small Nucleolar); 0 (RNA, U3 small nucleolar); 0.
. .

=> s tar and (nucleolar or nucleolus)
L7 83 TAR AND (NUCLEOLAR OR NUCLEOLUS)

=> s l7 and py<2001
3 FILES SEARCHED...
L8 31 L7 AND PY<2001

=> dup rem l8
PROCESSING COMPLETED FOR L8
L9 22 DUP REM L8 (9 DUPLICATES REMOVED)

=> d l9 ibib abs tot

L9 ANSWER 1 OF 22 USPATFULL on STN
ACCESSION NUMBER: 2000:109568 USPATFULL
TITLE: Antisense viruses and antisense-ribozyme viruses
INVENTOR(S): Hu, Wen, Honolulu, HI, United States
Wang, Jie, Honolulu, HI, United States
PATENT ASSIGNEE(S): Inpax, Inc., Honolulu, HI, United States (U.S. corporation)

	NUMBER	KIND	DATE	
PATENT INFORMATION:	US 6107062		20000822	<--
APPLICATION INFO.:	US 1992-921104		19920730	(7)

DOCUMENT TYPE: Utility
FILE SEGMENT: Granted
PRIMARY EXAMINER: Guzo, David
LEGAL REPRESENTATIVE: Nixon & Vanderhye P.C.
NUMBER OF CLAIMS: 11
EXEMPLARY CLAIM: 1
NUMBER OF DRAWINGS: 7 Drawing Figure(s); 7 Drawing Page(s)
LINE COUNT: 6292
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB Antisense viruses and antisense ribozyme viruses are disclosed. The novel artificial viruses, their synthesis and their use in preventing and treating viral infections are presented.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L9 ANSWER 2 OF 22 USPATFULL on STN
ACCESSION NUMBER: 2000:77225 USPATFULL
TITLE: Antisense modulation of Survivin expression
INVENTOR(S): Bennett, C. Frank, Carlsbad, CA, United States
Ackermann, Elizabeth J., Solana Beach, CA, United States
Swayze, Eric E., Carlsbad, CA, United States
Cowsert, Lex M., Carlsbad, CA, United States
PATENT ASSIGNEE(S): Isis Pharmaceuticals Inc., Carlsbad, CA, United States (U.S. corporation)

	NUMBER	KIND	DATE	
PATENT INFORMATION:	US 6077709		20000620	<--
APPLICATION INFO.:	US 1998-163162		19980929	(9)

DOCUMENT TYPE: Utility
FILE SEGMENT: Granted
PRIMARY EXAMINER: Brusca, John S.
ASSISTANT EXAMINER: McGarry, Sean
LEGAL REPRESENTATIVE: Law Offices of Jane Massey Licata
NUMBER OF CLAIMS: 20
EXEMPLARY CLAIM: 1
LINE COUNT: 1974
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB Antisense compounds, compositions and methods are provided for modulating the expression of Survivin. The compositions comprise antisense compounds, particularly antisense oligonucleotides, targeted to nucleic acids encoding Survivin. Methods of using these compounds for modulation of Survivin expression and for treatment of diseases associated with expression of Survivin are provided.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L9 ANSWER 3 OF 22 USPATFULL on STN
ACCESSION NUMBER: 2000:70964 USPATFULL
TITLE: Method of intracellular binding of target molecules
INVENTOR(S): Marasco, Wayne A., Wellesley, MA, United States
Haseltine, William A., Cambridge, MA, United States
PATENT ASSIGNEE(S): Dana-Farber Cancer Institute, Inc., Boston, MA, United States (U.S. corporation)

	NUMBER	KIND	DATE	
PATENT INFORMATION:	US 6072036		20000606	<--
APPLICATION INFO.:	US 1999-287145		19990406	(9)

RELATED APPLN. INFO.: Division of Ser. No. US 1995-438190, filed on 9 May 1995, now patented, Pat. No. US 5965371 which is a continuation of Ser. No. US 1993-45274, filed on 31 Mar 1993 which is a continuation-in-part of Ser. No. US 1992-916939, filed on 17 Jul 1992
DOCUMENT TYPE: Utility
FILE SEGMENT: Granted
PRIMARY EXAMINER: Stucker, Jeffrey
LEGAL REPRESENTATIVE: Eisenstein, Ronald I., Resnick, David S.Nixon Peabody LLP
NUMBER OF CLAIMS: 5
EXEMPLARY CLAIM: 1
NUMBER OF DRAWINGS: 19 Drawing Figure(s); 17 Drawing Page(s)
LINE COUNT: 2773
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB The present invention relates to a method by which one can target an undesired target molecule or target antigen, preferably a protein. The method comprises the intracellular expression of an antibody capable of

binding to the target. A DNA sequence is delivered to a cell, the DNA sequence contains a sufficient number of nucleotides coding for the portion of an antibody capable of binding to the target operably linked to a promoter that will permit expression of the antibody in the cell(s) of interest. The antibody is then expressed intracellularly and binds to the target, thereby disrupting the target from its normal actions.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L9 ANSWER 4 OF 22 USPATFULL on STN

ACCESSION NUMBER:	2000:24460 USPATFULL			
TITLE:	Antisense modulation of RhoC expression			
INVENTOR(S):	Cowsert, Lex M., Carlsbad, CA, United States			
PATENT ASSIGNEE(S):	Isis Pharmaceuticals, Inc., Carlsbad, CA, United States (U.S. corporation)			
	NUMBER	KIND	DATE	
PATENT INFORMATION:	US 6030786		20000229	<--
APPLICATION INFO.:	US 1998-156807		19980918	(9)
DOCUMENT TYPE:	Utility			
FILE SEGMENT:	Granted			
PRIMARY EXAMINER:	Elliott, George C.			
ASSISTANT EXAMINER:	Wang, Andrew			
LEGAL REPRESENTATIVE:	Law Offices of Jane Massey Licata			
NUMBER OF CLAIMS:	12			
EXEMPLARY CLAIM:	1			
LINE COUNT:	1956			

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

AB Antisense compounds, compositions and methods are provided for modulating the expression of RhoC. The compositions comprise antisense compounds, particularly antisense oligonucleotides, targeted to nucleic acids encoding RhoC. Methods of using these compounds for modulation of RhoC expression and for treatment of diseases associated with expression of RhoC are provided.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L9 ANSWER 5 OF 22 USPATFULL on STN

ACCESSION NUMBER:	2000:9723 USPATFULL			
TITLE:	Unique nucleotide and amino acid sequence and uses thereof			
INVENTOR(S):	Summers, Max D., Bryan, TX, United States Braunagel, Sharon C., Bryan, TX, United States Hong, Tao, Bryan, TX, United States			
PATENT ASSIGNEE(S):	The Texas A & M University System, College Station, TX, United States (U.S. corporation)			
	NUMBER	KIND	DATE	
PATENT INFORMATION:	US 6017734		20000125	<--
APPLICATION INFO.:	US 1997-792832		19970130	(8)
RELATED APPLN. INFO.:	Continuation-in-part of Ser. No. US 1996-678435, filed on 3 Jul 1996, now abandoned			
	NUMBER		DATE	
PRIORITY INFORMATION:	US 1995-955P		19950707	(60)
DOCUMENT TYPE:	Utility			
FILE SEGMENT:	Granted			
PRIMARY EXAMINER:	Elliott, George C.			
ASSISTANT EXAMINER:	Schwartzman, Robert			
LEGAL REPRESENTATIVE:	Arnold, White & Durkee			
NUMBER OF CLAIMS:	56			
EXEMPLARY CLAIM:	1			

NUMBER OF DRAWINGS: 47 Drawing Figure(s); 24 Drawing Page(s)
LINE COUNT: 7846
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB Provided are hydrophobic targeting sequences, which may serve to target heterologous proteins to a variety of cellular membranes. In particular, the structural components of the nuclear envelope, or those components which become nucleus-associated, may be targeted with the sequences provided. Also provided are methods of targeting heterologous proteins to particular membranes, and the use of these targeted proteins in therapeutic, diagnostic and insecticidal applications.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L9 ANSWER 6 OF 22 CAPLUS COPYRIGHT 2003 ACS on STN
ACCESSION NUMBER: 2000:717215 CAPLUS
DOCUMENT NUMBER: 134:176440
TITLE: Identification of **nucleolar** protein No55 as a tumour-associated autoantigen in patients with prostate cancer
AUTHOR(S): Fossa, A.; Siebert, R.; Aasheim, H.-C.; Maelandsmo, G. M.; Berner, A.; Fossa, S. D.; Paus, E.; Smeland, E. B.; Gaudernack, G.
CORPORATE SOURCE: Department of Immunology, The Norwegian Radium Hospital, Oslo, 0310, Norway
SOURCE: British Journal of Cancer (**2000**), 83(6), 743-749
CODEN: BJCAAI; ISSN: 0007-0920
PUBLISHER: Harcourt Publishers Ltd.
DOCUMENT TYPE: Journal
LANGUAGE: English
AB Four different genes were identified by immunoscreening of a cDNA expression library from the human prostate cancer cell line DU145 with allogeneic sera from four prostate cancer patients. A cDNA encoding the **nucleolar** protein No55 was further analyzed and shown to be expressed at the mRNA level in several normal tissues, including ovaries, pancreas and prostate and in human prostate cancer cell lines PC-3, PC-3m and LNCaP. By reverse transcriptase/polymerase chain reaction, expression of No55 was several-fold higher in two out of nine prostate cancer primary tumors and two out of two metastatic lesions, compared to normal prostate tissue. Antibodies to No55 were detected in sera from seven out of 47 prostate cancer patients but not in sera from 20 healthy male controls. Sequence anal. of the No55 open reading frame from normal and tumor tissues revealed no tumor-specific mutations. The No55 gene was located to chromosome 17q21, a region reported to be partially deleted in prostate cancer. Considering the immunogenicity of the No55 protein in the tumor host, the expression profile and chromosomal localization of the corresponding gene, studies evaluating No55 as a potential antigen for immunol. studies in prostate cancer may be warranted.
REFERENCE COUNT: 32 THERE ARE 32 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L9 ANSWER 7 OF 22 USPATFULL on STN
ACCESSION NUMBER: 1999:159824 USPATFULL
TITLE: Helper virus-free herpesvirus vector packaging system
INVENTOR(S): Fraefel, Cornel, Brookline, MA, United States
Geller, Alfred I., Boston, MA, United States
Lim, Filip, Brookline, MA, United States
PATENT ASSIGNEE(S): Children's Medical Center Corporation, Boston, MA, United States (U.S. corporation)

	NUMBER	KIND	DATE	
PATENT INFORMATION:	US 5998208		19991207	<--
APPLICATION INFO.:	US 1998-9925		19980121 (9)	

RELATED APPLN. INFO.: Continuation of Ser. No. US 1995-508088, filed on 26

Jul 1995, now patented, Pat. No. US 5851826
DOCUMENT TYPE: Utility
FILE SEGMENT: Granted
PRIMARY EXAMINER: Degen, Nancy
ASSISTANT EXAMINER: McGarry, Sean
LEGAL REPRESENTATIVE: Resnick, David S., Eisenstein, Ronald I.Nixon Peabody LLP
NUMBER OF CLAIMS: 9
EXEMPLARY CLAIM: 1
NUMBER OF DRAWINGS: 20 Drawing Figure(s); 8 Drawing Page(s)
LINE COUNT: 1169
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB We have now discovered a helper virus free herpesvirus packaging system. This system can be used to package a wide range of desired nucleotide segments, preferably a DNA segment, into an empty herpesvirus particle because of the large genomes of herpesviruses. Preferably, the herpesvirus is an alpha herpesvirus. More preferably, the herpesvirus is an alpha herpesvirus such as Varicella-Zoster virus, pseudorabies virus, or a herpes simplex virus such as HSV-1 or HSV-2. Another preferred herpesvirus is Epstein-Barr virus.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L9 ANSWER 8 OF 22 USPATFULL on STN
ACCESSION NUMBER: 1999:150917 USPATFULL
TITLE: Screening methods in eucaryotic cells
INVENTOR(S): Frankel, Allan, Mill Valley, CA, United States
Tan, Ruoying, San Francisco, CA, United States
PATENT ASSIGNEE(S): Reagents of the University of California, Oakland, CA, United States (U.S. corporation)

	NUMBER	KIND	DATE	
PATENT INFORMATION:	US 5989814		19991123	<--
APPLICATION INFO.:	US 1997-847176		19970401	(8)

DOCUMENT TYPE: Utility
FILE SEGMENT: Granted
PRIMARY EXAMINER: Fredman, Jeffrey
LEGAL REPRESENTATIVE: Townsend & Townsend & Crew
NUMBER OF CLAIMS: 33
EXEMPLARY CLAIM: 1
NUMBER OF DRAWINGS: 13 Drawing Figure(s); 10 Drawing Page(s)
LINE COUNT: 2001
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB The invention provides new methods for screening libraries of peptides and other compounds for a desired property in eucaryotic cells. The methods are premised, in part, on the unexpected observation that the contents of procaryotic or lower eucaryotic cells, such as yeast, can be transferred to recipient eucaryotic cells in an essentially clonal manner by protoplast fusion of the respective cells. Applications of the methods include screening peptides in eucaryotic cells substantially incapable of episomal replication of transferred nucleic acid fragments; screening in eucaryotic cells peptides or secondary metabolites produced in procaryotic cells; and screening a library of peptides for capacity to bind a selected RNA in eucaryotic cells.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L9 ANSWER 9 OF 22 USPATFULL on STN
ACCESSION NUMBER: 1999:124707 USPATFULL
TITLE: Method of intracellular binding of target molecules
INVENTOR(S): Marasco, Wayne A., Wellesley, MA, United States
Haseltine, William A., Cambridge, MA, United States
PATENT ASSIGNEE(S): Dana-Farber Cancer Institute, Boston, MA, United States (U.S. corporation)

	NUMBER	KIND	DATE	
PATENT INFORMATION:	US 5965371		19991012	<--
APPLICATION INFO.:	US 1995-438190		19950509 (8)	

RELATED APPLN. INFO.: Continuation of Ser. No. US 1993-45274, filed on 31 Mar 1993 which is a continuation-in-part of Ser. No. US 1992-916939, filed on 17 Jul 1992, now abandoned
DOCUMENT TYPE: Utility
FILE SEGMENT: Granted
PRIMARY EXAMINER: Stucker, Jeffrey
LEGAL REPRESENTATIVE: Eisenstein, Ronald I., Conlin, David G., Resnick, David S.
NUMBER OF CLAIMS: 101
EXEMPLARY CLAIM: 1
NUMBER OF DRAWINGS: 33 Drawing Figure(s); 17 Drawing Page(s)
LINE COUNT: 3086
CAS INDEXING IS AVAILABLE FOR THIS PATENT.

AB The present invention relates to a method by which one can target an undesired target molecule or target antigen, preferably a protein. The method comprises the intracellular expression of an antibody capable of binding to the target. A DNA sequence is delivered to a cell, the DNA sequence contains a sufficient number of nucleotides coding for the portion of an antibody capable of binding to the target operably linked to a promoter that will permit expression of the antibody in the cell(s) of interest. The antibody is then expressed intracellularly and binds to the target, thereby disrupting the target from its normal actions.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L9 ANSWER 10 OF 22 USPATFULL on STN
ACCESSION NUMBER: 1999:117339 USPATFULL
TITLE: Chimeric antiviral agents comprising Rev binding nucleic acids and trans-acting ribozymes, and molecules encoding them
INVENTOR(S): Kraus, Gunter, Miami, FL, United States
Wong-Staal, Flossie, San Diego, CA, United States
Yu, Mang, San Diego, CA, United States
Yamada, Osamu, Kobe, Japan
PATENT ASSIGNEE(S): The Regents of the University of California, Oakland, CA, United States (U.S. corporation)

	NUMBER	KIND	DATE	
PATENT INFORMATION:	US 5958768		19990928	<--
APPLICATION INFO.:	US 1996-697324		19960823 (8)	

	NUMBER	DATE
PRIORITY INFORMATION:	US 1995-2793P	19950825 (60)

DOCUMENT TYPE: Utility
FILE SEGMENT: Granted
PRIMARY EXAMINER: Smith, Lynette F.
ASSISTANT EXAMINER: Nelson, Amy J.
LEGAL REPRESENTATIVE: Townsend and Townsend and Crew LLP
NUMBER OF CLAIMS: 25
EXEMPLARY CLAIM: 1,21
NUMBER OF DRAWINGS: 18 Drawing Figure(s); 7 Drawing Page(s)
LINE COUNT: 2347
CAS INDEXING IS AVAILABLE FOR THIS PATENT.

AB Methods and compositions for the treatment and diagnosis of infections of Rev-binding primate lentiviruses are provided. These methods and compositions utilize the ability of Rev binding nucleic acids such as the SLII sequence from the HIV-1 Rev response element (RRE) to target therapeutic agents to the same sub-cellular location as primate

lentiviruses which contain RRE sequences. In particular, the invention provides trans-acting ribozymes comprising Rev-binding nucleic acids less toxic than a full-length RRE, and molecules encoding them. The use of the compositions of the invention as components of diagnostic assays, as prophylactic reagents, and in vectors is also described.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L9 ANSWER 11 OF 22 USPATFULL on STN

ACCESSION NUMBER:	1999:81758 USPATFULL			
TITLE:	Non-activated receptor complex proteins and uses thereof			
INVENTOR(S):	Davis, Roger J., Princeton, MA, United States Galcheva-Gargova, Zoya, Worcester, MA, United States			
PATENT ASSIGNEE(S):	University of Massachusetts, Boston, MA, United States (U.S. corporation)			
	NUMBER	KIND	DATE	
PATENT INFORMATION:	US 5925566		19990720	<--
APPLICATION INFO.:	US 1997-870518		19970606	(8)
	NUMBER		DATE	
PRIORITY INFORMATION:	US 1996-19219P		19960606 (60)	
DOCUMENT TYPE:	Utility			
FILE SEGMENT:	Granted			
PRIMARY EXAMINER:	Campell, Bruce R.			
ASSISTANT EXAMINER:	Nguyen, Dave Trong			
LEGAL REPRESENTATIVE:	Fish & Richardson, P.C.			
NUMBER OF CLAIMS:	23			
EXEMPLARY CLAIM:	1			
NUMBER OF DRAWINGS:	22 Drawing Figure(s); 18 Drawing Page(s)			
LINE COUNT:	2438			

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

AB The invention features a substantially pure ZPR1 polypeptide. For example, a ZPR1 polypeptide that specifically binds to a non-activated membrane-bound receptor (e.g., EGF or PDGF receptors) and specifically binds small **nucleolar** RNAs (e.g., U3). ZPR1 polypeptides can be isolated from any eukaryote, including mammals (e.g. rodents and humans) and fungi (e.g., S. cerevisiae and S. pombe).

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L9 ANSWER 12 OF 22 USPATFULL on STN

ACCESSION NUMBER:	1998:159761 USPATFULL			
TITLE:	Method of intracellular binding of target molecules			
INVENTOR(S):	Marasco, Wayne A., Wellesley, MA, United States Haseltine, William A., Rockville, MD, United States			
PATENT ASSIGNEE(S):	Dana-Farber Cancer Institute, Boston, MA, United States (U.S. corporation)			
	NUMBER	KIND	DATE	
PATENT INFORMATION:	US 5851829		19981222	<--
	WO 9402610		19940203	<--
APPLICATION INFO.:	US 1995-373190		19950330	(8)
	WO 1993-US6735		19930716	
			19950330	PCT 371 date
			19950330	PCT 102(e) date
DOCUMENT TYPE:	Utility			
FILE SEGMENT:	Granted			
PRIMARY EXAMINER:	Stucker, Jeffrey			
LEGAL REPRESENTATIVE:	Conlin, David G., Eisenstein, Ronald I., Resnick, David S.			

NUMBER OF CLAIMS: 62
EXEMPLARY CLAIM: 1
NUMBER OF DRAWINGS: 34 Drawing Figure(s); 19 Drawing Page(s)
LINE COUNT: 3209
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB The present invention relates to a method by which one can target an undesired target molecule or target antigen, preferably a protein. The method comprises the intracellular expression of an antibody capable of binding to the target. A DNA sequence is delivered to a cell, the DNA sequence contains a sufficient number of nucleotides coding for the portion of an antibody capable of binding to the target operably linked to a promoter that will permit expression of the antibody in the cell(s) of interest. The antibody is then expressed intracellularly and binds to the target, thereby disrupting the target from its normal actions.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L9 ANSWER 13 OF 22 USPATFULL on STN
ACCESSION NUMBER: 1998:159758 USPATFULL
TITLE: Helper virus-free herpesvirus vector packaging system
INVENTOR(S): Fraefel, Cornel, Brookline, MA, United States
Geller, Alfred I., Boston, MA, United States
Lim, Filip, Brookline, MA, United States
PATENT ASSIGNEE(S): Children's Medical Center Corporation, Boston, MA, United States (U.S. corporation)

	NUMBER	KIND	DATE	
PATENT INFORMATION:	US 5851826		19981222	<--
APPLICATION INFO.:	US 1995-508088		19950726	(8)

DOCUMENT TYPE: Utility
FILE SEGMENT: Granted
PRIMARY EXAMINER: Elliott, George C.
ASSISTANT EXAMINER: Garry, Sean M.
LEGAL REPRESENTATIVE: Conlin, David G., Resnick, David S.Dike, Bronstein, Roberts & Cushman, LLP
NUMBER OF CLAIMS: 19
EXEMPLARY CLAIM: 1
NUMBER OF DRAWINGS: 20 Drawing Figure(s); 8 Drawing Page(s)
LINE COUNT: 1198
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB We have now discovered a helper virus free herpesvirus packaging system. This system can be used to package a wide range of desired nucleotide segments, preferably a DNA segment, into an empty herpesvirus particle because of the large genomes of herpesviruses. Preferably, the herpesvirus is an alpha herpesvirus. More preferably, the herpesvirus is an alpha herpesvirus such as Varicella-Zoster virus, pseudorabies virus, or a herpes simplex virus such as HSV-1 or HSV-2. Another preferred herpesvirus is Epstein-Barr virus.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L9 ANSWER 14 OF 22 MEDLINE on STN DUPLICATE 1
ACCESSION NUMBER: 97360029 MEDLINE
DOCUMENT NUMBER: 97360029 PubMed ID: 9217057
TITLE: Bovine immunodeficiency virus tat gene: cloning of two distinct cDNAs and identification, characterization, and immunolocalization of the tat gene products.
AUTHOR: Fong S E; Greenwood J D; Williamson J C; Derse D; Pallansch L A; Copeland T; Rasmussen L; Mentzer A; Nagashima K; Tobin G; Gonda M A
CORPORATE SOURCE: Laboratory of Cell and Molecular Structure, SAIC Frederick, NCI-Frederick Cancer Research and Development Center, Maryland 21702-1201, USA.. Fong@mail.ncifcrf.gov
SOURCE: VIROLOGY, **(1997 Jul 7)** 233 (2) 339-57.

Journal code: 0110674. ISSN: 0042-6822.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals; AIDS
ENTRY MONTH: 199708
ENTRY DATE: Entered STN: 19970825
Last Updated on STN: 19970825
Entered Medline: 19970813

AB cDNAs encoding the bovine immunodeficiency virus (BIV) transactivator gene (tat) were cloned from virally infected cells and characterized. BIV expresses two distinct tat mRNAs composed of three exons that are derived by alternative splicing. The BIV tat mRNA splice variants encode Tat proteins of 103 (Tat103) and 108 (Tat108) amino acids. The Tat103 coding region is specified only by exon 2, while that of Tat108 is specified by a truncated exon 2 and the first 30 nt of exon 3. Thus, the first 98 amino acids of each Tat are identical, and have amino terminal, cysteine-rich, conserved core, basic, and carboxyl-terminal domains similar to Tats encoded by primate lentiviruses. BIV-infected bovine cells express a 14-kDa phosphorylated Tat protein identical in size to recombinant Tat expressed in bacteria. BIV Tat was shown to localize exclusively in the nucleoli of virally infected and Tat-expressing cells. Reporter gene assays indicated that Tat103 and Tat108 can strongly transactivate the BIV long terminal repeat (LTR) in virally permissive canine Cf2Th and nonpermissive HeLa and mouse NIH 3T3 cells, but not in permissive lapine EREp cells. However, an intact BIV tat gene is required for viral replication in both Cf2Th and EREp cells. Strong LTR activation by BIV Tat requires a **TAR** (transactivation responsive) element delimited by viral nt +1 to +31 and the Tat basic domain. BIV Tat strongly cross-transactivates the HIV-1 LTR in a **TAR**-dependent manner in Cf2Th, but not in EREp, HeLa, or NIH 3T3 cells. In contrast, strong, **TAR**-dependent cross-transactivation of the BIV LTR by HIV-1 Tat could not be demonstrated in any of these cell types. In Cf2Th cells Tat108 effects a moderately stronger transactivation of the BIV LTR than Tat103, indicative of a functional difference in BIV Tat proteins encoded by the mRNA splice variants. The present studies demonstrate that BIV Tat parallels the primate lentiviral Tats in structure and biochemistry but is not interchangeable with the latter.

L9 ANSWER 15 OF 22 CAPLUS COPYRIGHT 2003 ACS on STN
ACCESSION NUMBER: 1997:493990 CAPLUS
DOCUMENT NUMBER: 127:188410
TITLE: Xlrbpa, a double-stranded RNA-binding protein associated with ribosomes and heterogeneous nuclear RNPs
AUTHOR(S): Eckmann, Christian R.; Jantsch, Michael F.
CORPORATE SOURCE: Department of Cytology and Genetics, Institute of Botany, University of Vienna, Vienna, A-1030, Austria
SOURCE: Journal of Cell Biology (**1997**), 138(2), 239-253
CODEN: JCLBA3; ISSN: 0021-9525
PUBLISHER: Rockefeller University Press
DOCUMENT TYPE: Journal
LANGUAGE: English

AB The authors have cloned and characterized Xlrbpa, a double-stranded RNA-binding protein from Xenopus laevis. Xlrbpa is a protein of 33 kDa and contains three tandemly arranged, double-stranded RNA-binding domains (dsRBDs) that bind exclusively to double-stranded RNA in vitro, but fail to bind either single-stranded RNA or DNA. Sequence data and the overall organization of the protein suggest that Xlrbpa is the Xenopus homolog of human **TAR**-RNA binding protein (TRBP), a protein isolated by its ability to bind to human immunodeficiency virus (HIV) **TAR**-RNA. In transfection assays, TRBP has also been shown to inhibit the interferon-induced protein kinase PKR possibly by direct phys. interaction. To det. the function of Xlrbpa and its human homolog, the

authors studied the expression and intracellular distribution of the two proteins. Xlrbpa is ubiquitously expressed with marked quant. differences amongst all tissues. Xlrbpa and human TRBP can be detected in the cytoplasm and nucleus by immunofluorescence staining and Western blotting. Sedimentation gradient analyses and immunopptn. expts. suggest an assocn. of cytoplasmic Xlrbpa with ribosomes. In contrast, a control construct contg. two dsRBDs fails to assoc. with ribosomes in microinjected Xenopus oocytes. Nuclear staining of Xenopus lampbrush chromosome prepns. showed the assocn. of the protein with nucleoli, again indicating an assocn. of the protein with rRNAs. Addnl., Xlrbpa could be located on lampbrush chromosomes and in snurposomes. Immunopptns. of nuclear exts. demonstrated the presence of the protein in heterogeneous nuclear (hn) RNP particles, but not in small nuclear RNPs, explaining the chromosomal localization of the protein. It thus appears that Xlrbpa is a general double-stranded RNA-binding protein which is assocd. with the majority of cellular RNAs, rRNAs, and hnRNAs either alone or as part of an hnRNP complex.

L9 ANSWER 16 OF 22 USPATFULL on STN
ACCESSION NUMBER: 95:47839 USPATFULL
TITLE: Methods and compositions for diagnosing HTLV-I associated myelopathy and adult T-cell leukemia
INVENTOR(S): Rudolph, Donna L., Tucker, GA, United States
Lal, Renu B., Atlanta, GA, United States
PATENT ASSIGNEE(S): The United States of America as represented by the Department of Health and Human Services, Washington, DC, United States (U.S. government)

	NUMBER	KIND	DATE		
PATENT INFORMATION:	US 5420244		19950530		<--
APPLICATION INFO.:	US 1993-103742		19930806	(8)	

DOCUMENT TYPE: Utility
FILE SEGMENT: Granted
PRIMARY EXAMINER: Nucker, Christine M.
ASSISTANT EXAMINER: Stucker, Jeffrey
LEGAL REPRESENTATIVE: Needle & Rosenberg
NUMBER OF CLAIMS: 8
EXEMPLARY CLAIM: 1
LINE COUNT: 1369
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB The invention provides antigenic peptides derived from immunodominant epitopes of the HTLV-I tax or rex proteins that are immunoreactive with antibodies associated with disease in HTLV-I infected subjects. More specifically, the invention provides antigenic peptides consisting essentially of the amino acid sequences defined in the sequence listing by SEQ ID NOs: 1, 2, 3, 4, 5, 6, 7 and 8 and antigenic fragments thereof. The invention provides a method of diagnosing HTLV-I associated myelopathy (HAM) or a predisposition thereto, comprising the steps of: (a) contacting an antibody containing sample from the subject with a detectable amount of a peptide of the invention or an antigenic fragment thereof; and (b) detecting the reaction of the peptide with an antibody in the sample, the reaction indicating HTLV-I associated myelopathy or a predisposition thereto. The invention also provides a method of diagnosing adult T-cell leukemia or a predisposition thereto in a subject, comprising the steps of: (a) contacting an antibody containing sample from the subject with a detectable amount of the peptide of SEQ ID NO: 1; and (b) detecting the reaction of the peptide with an antibody in the sample, the reaction indicating adult T-cell leukemia or predisposition thereto.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L9 ANSWER 17 OF 22 CAPLUS COPYRIGHT 2003 ACS on STN
ACCESSION NUMBER: 1993:401784 CAPLUS

DOCUMENT NUMBER: 119:1784
TITLE: Cellular factors in HIV transactivation
AUTHOR(S): Wong-Staal, Flossie
CORPORATE SOURCE: Univ. California, San Diego, La Jolla, CA, USA
SOURCE: AIDS Research Reviews (**1992**), 2, 29-40
CODEN: ARRVEZ; ISSN: 1056-1080
DOCUMENT TYPE: Journal; General Review
LANGUAGE: English

AB A review with 60 refs. RNA-protein interaction is a primary mode of regulation of HIV gene expression and is likely to be important for eukaryotic gene regulation in general. However, relatively little is known about such as mechanism. Tat and Rev functions were studied to examine cellular factors that participate in these transactivation pathways. A single protein of 50-55 kDa has been identified so far that binds specifically to the Rev response element (RRE) in the vicinity of the Rev binding site. This protein, NFRRE, appears to be important for Rev function and, by implication, may be involved in mRNA processing and transport. Rev, on the other hand, binds to the **nucleolar** B23 protein, which may contribute to shuttling and storage of Rev. **TAR** binds to a no. of cellular proteins, at least two of which have been shown to contribute to the overall activity of Tat. The multiplicity of cellular proteins interacting with **TAR** may account for different facets of Tat function, which have been proposed to include both transcriptional initiation and elongation. Elucidation of the function of these proteins would help in the identification of specific strategies for viral intervention as well as greatly expand our existing knowledge on gene regulation.

L9 ANSWER 18 OF 22 CAPLUS COPYRIGHT 2003 ACS on STN DUPLICATE 2
ACCESSION NUMBER: 1991:487793 CAPLUS
DOCUMENT NUMBER: 115:87793
TITLE: Heterologous basic domain substitutions in the HIV-1 Tat protein reveal an arginine-rich motif required for transactivation
AUTHOR(S): Subramanian, T.; Govindarajan, R.; Chinnadurai, G.
CORPORATE SOURCE: Inst. Mol. Virol., St. Louis Univ., St. Louis, MO, 63110, USA
SOURCE: EMBO Journal (**1991**), 10(8), 2311-18
CODEN: EMJODG; ISSN: 0261-4189
DOCUMENT TYPE: Journal
LANGUAGE: English

AB The Tat protein coded by HIV-1 is a unique eukaryotic transactivator. It activates gene expression from the viral LTR by its interaction with a nascent RNA element (**TAR**) located at the 5' end of all HIV-1 transcripts. Tat appears to bind to its target structure in a highly sequence-specific manner. The **TAR**-binding activity of Tat has been localized in an Arg-rich basic domain located between residues 49 and 57 of the Tat protein. Domain substitution studies with heterologous basic domains which are also implicated in RNA binding was carried out. It is reported that a 19 or a 12 amino acid region from the N-terminus of HTLV-I Rex can functionally substitute for the Tat basic domain. In contrast, the Arg-rich domains of the N gene products of bacteriophages .lambda. and 21 do not functionally substitute for the Tat basic domain. The pos. and neg. effects of various domain substitutions have facilitated identification of a consensus sequence (Arg/Lys-X-X-Arg-Arg-X-Arg-Arg) in the basic domain required for Tat activity. Conservation of the functionally inactive basic domain of the .lambda. N protein to the consensus motif restored the transactivation function of the Tat-N chimeric protein. Similarly, the Rex basic domain contg. scrambled sequences unrelated or partially related to the consensus motif were either totally defective in transactivation or exhibited reduced activity. The results further suggest that the activity of the core Arg motif may be enhanced by the presence of glutamine or asparagine within the basic domain. It has been shown that the basic doamin is required for targeting Tat to the nucleus and **nucleolus**. The chimeric Tat proteins

contg. the basic domains of Rex and the N proteins are also localized in the nuclear/**nucleolar** region. Since the functionally inactive Tat-N chimeric proteins are still efficiently targeted to the nuclear/**nucleolar** compartments, the results suggest that nuclear/**nucleolar** localization alone is not sufficient for transactivation and demonstrate a direct role for the Arg motif in Tat function other than that required for nuclear/**nucleolar** localization.

L9 ANSWER 19 OF 22 MEDLINE on STN DUPLICATE 3
ACCESSION NUMBER: 90311346 MEDLINE
DOCUMENT NUMBER: 90311346 PubMed ID: 2195547
TITLE: A transdominant tat mutant that inhibits tat-induced gene expression from the human immunodeficiency virus long terminal repeat.
AUTHOR: Pearson L; Garcia J; Wu F; Modesti N; Nelson J; Gaynor R
CORPORATE SOURCE: Department of Medicine, University of California-Los Angeles School of Medicine 90024.
SOURCE: PROCEEDINGS OF THE NATIONAL ACADEMY OF SCIENCES OF THE UNITED STATES OF AMERICA, **(1990 Jul)** 87 (13) 5079-83.
Journal code: 7505876. ISSN: 0027-8424.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals; AIDS
ENTRY MONTH: 199008
ENTRY DATE: Entered STN: 19900921
Last Updated on STN: 19970203
Entered Medline: 19900815

AB Regulation of human immunodeficiency virus (HIV) gene expression is dependent on specific regulatory regions in the long terminal repeat. These regions include the enhancer, SP1, "TATA," and trans-activating (**TAR**) regions. In addition, viral regulatory proteins such as tat and rev are important in regulating HIV gene expression. The mechanism of tat activation remains the subject of investigation, but effects at both transcriptional and posttranscriptional levels seem likely. Previous mutagenesis of the tat protein revealed that the amino terminus, the cysteine-rich domain, and the basic domain were all required for complete tat activation. Mutants of other viral trans-acting regulatory proteins, including E1A, tax, and VM65, have been identified that were capable of antagonizing the activity of their corresponding wild-type proteins. We wished to determine whether mutants of the tat protein could be identified that exhibited a similar phenotype. One mutant (delta tat) that truncated the basic domain of tat resulted in a transdominant phenotype inhibiting tat-induced gene expression of the HIV long terminal repeat but not other viral promoters. This mutant exhibited its maximal phenotype in cotransfection experiments when present in an 8- to 30-fold molar excess over the wild-type tat gene. Trans-activation of the HIV long terminal repeat by delta tat was very defective at the DNA concentrations used in these experiments. RNase protection analysis indicated that this mutant decreased tat-induced steady-state mRNA levels of the HIV long terminal repeat. Second-site mutations of the delta tat gene in either the amino terminus or cysteine region eliminated the transdominant phenotype. In contrast to tat, which was localized predominantly to the **nucleolus**, delta tat was present in both the nucleus and cytoplasm, suggesting that it may inhibit tat function by preventing **nucleolar** localization. Transdominant mutants of tat may have a role in potentially inhibiting HIV gene expression.

L9 ANSWER 20 OF 22 MEDLINE on STN DUPLICATE 4
ACCESSION NUMBER: 91032988 MEDLINE
DOCUMENT NUMBER: 91032988 PubMed ID: 2227414
TITLE: A bulge structure in HIV-1 **TAR** RNA is required for Tat binding and Tat-mediated trans-activation.
AUTHOR: Roy S; Delling U; Chen C H; Rosen C A; Sonenberg N

CORPORATE SOURCE: Department of Biochemistry, McGill University, Montreal, Quebec, Canada.
SOURCE: GENES AND DEVELOPMENT, **(1990 Aug)** 4 (8) 1365-73.
Journal code: 8711660. ISSN: 0890-9369.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals; AIDS
ENTRY MONTH: 199012
ENTRY DATE: Entered STN: 19910208
Last Updated on STN: 19970203
Entered Medline: 19901219

AB The Tat protein of human immunodeficiency virus type 1 (HIV-1) trans-activates viral gene expression and is obligatory for virus replication. Tat function is mediated through a sequence termed **TAR** that comprises part of the 5'-noncoding region of all HIV-1 mRNAs. This region forms a stable stem-loop structure in vitro. Recent evidence indicates that Tat binds directly to the **TAR** RNA sequence, and this binding is independent of the nucleotide sequence in the loop but dependent on the integrity of the upper stem. We used the electrophoretic mobility-shift assay to identify the sequence and structure specificity of this interaction and its correlation with Tat trans-activation. We show that a 3-nucleotide bulge structure (positions +23 to +25) in **TAR** RNA is important for both Tat interaction with **TAR** RNA and Tat-mediated trans-activation of gene expression. Single base substitutions at position +23 that impair Tat-mediated trans-activation in vivo also reduce binding of Tat to **TAR** in vitro, suggesting that the first uridine residue in the bulge is the critical base for both functions. In contrast, mutations in the loop (positions +31 to +34) and the stem (positions +9 to +12 and +49 to +52), which reduce Tat-mediated trans-activation, had no effect on Tat binding. We also show that a Tat peptide that includes the basic region required for **nucleolar** localization binds to **TAR** RNA with the same specificity as the full-length protein. We conclude that Tat binding to **TAR** is necessary but not sufficient by itself to account for trans-activation.

L9 ANSWER 21 OF 22 CAPLUS COPYRIGHT 2003 ACS on STN
ACCESSION NUMBER: 1956:21176 CAPLUS
DOCUMENT NUMBER: 50:21176
ORIGINAL REFERENCE NO.: 50:4373b-d
TITLE: Early cytologic changes produced by carcinogens
AUTHOR(S): Cooper, Norman S.
CORPORATE SOURCE: New York Univ.-Belluvue Med. Center, New York, NY
SOURCE: Bulletin of the New York Academy of Medicine (**1956**), 32, 79-80
CODEN: BNYMAM; ISSN: 0028-7091
DOCUMENT TYPE: Journal
LANGUAGE: Unavailable

AB Enlargement of mouse epidermal cell nucleoli was used as a criterion of the carcinogenicity of a substance painted on the skin of the animal. U.S.P. white mineral oil caused enlargement, therefore only materials giving enlargements greater than this were considered pos. Cigaret-smoke condensate (50% in acetone) was pos.; 50% coal **tar** in acetone gave even greater enlargement. The greatest enlargement was given by 0.5% 3,4-benzopyrene in acetone. A 50% soln. of turpentine in acetone produced **nucleolar** enlargement only slightly less than that resulting from tobacco **tar**.

L9 ANSWER 22 OF 22 CAPLUS COPYRIGHT 2003 ACS on STN
ACCESSION NUMBER: 1928:18650 CAPLUS
DOCUMENT NUMBER: 22:18650
ORIGINAL REFERENCE NO.: 22:2199b-e
TITLE: Studies in the microchemistry of the cell. I. The chromatin content of normal and malignant cells as

demonstrated by Feulgen's "Nuclealreaktion"
AUTHOR(S): Ludford, R. J.
SOURCE: Proc. Roy. Soc. (London) (**1928**), B102, 397-406
DOCUMENT TYPE: Journal
LANGUAGE: Unavailable
AB The following results were obtained by use of this reaction. In the rat and the mouse, the chromatin content of the nucleus does not increase during oogenesis, and no chromatin is extruded into the cytoplasm when the germinal vesicle breaks down to form the chromosomes. Chromatin is not present in either the oxyphilic or the basophilic nucleoli of the oocyte of the mollusk, Limnoea stagnalis. The sperm heads of all 3 species yield a faint reaction when the chromosomes are stretched; but the reaction becomes progressively more marked as condensation occurs in spermatogenesis. The chromosomes apparently contain other substances besides chromatin. Extrusion of chromatin does not occur in the epithelial cells of the epididymis. Shrinkage of the nuclei of gland cells after secretion (e. g., in the adrenal medulla after exposure to cold) is apparently due to the loss of some substance other than chromatin. No relationship is found between the chromatin content of a tumor cell nucleus and the rate of growth of the tumor. In a **tar** tumor and the surrounding skin, the chromatin content is apparently the same in the normal and the malignant cells. During cellular degeneration of tumors, their nuclei become shrunken and the chromatin runs together. In tumors, the chromosomes stain intensely, and are separate from the **nucleolus** during the prophase of nitosis. Nuclear extrusions, which are well marked in some tumors, consist of **nucleolar** material and not chromatin. **Nucleolar** extrusions are the chief source of keratohyalin during cornification. While the same amt. of chromatin may be present in both large and small nuclei, yet giant nuclei contain large masses of chromatin.

```
=> s tar and decoy and (nucleolus or nucleolar)
L10         18 TAR AND DECOY AND (NUCLEOLUS OR NUCLEOLAR)

=> dup rem l10
PROCESSING COMPLETED FOR L10
L11          12 DUP REM L10 (6 DUPLICATES REMOVED)

=> d l11 kwic 1-20

L11  ANSWER 1 OF 12  USPATFULL on STN
DETD   . . . the human immunodeficiency viruses (HIV). Tat is a
       transcriptional regulator protein that acts by binding to the
       transactivating response sequence (TAR) RNA element and
       activates transcription Initiation and/or elongation from the LTR
       promoter. HIV cannot replicate without tat, but the chemical. . .
DETD   . . . falciparum and influenza virus isolates. For example,
       Plasmodium falciparum contains several partial Replikins, referred to
       herein as "Replikin decoys." These decoy structures contain an
       abundance of lysine residues, but lack the histidine required of
       Replikin structures. Specifically, these decoys contain many. . .
       residues apart in overlapping fashion, similar to the true malaria
       recognins but without histidine residues. It is believed that the
       decoy structure maximizes the chances that an anti-malarial
       antibody or other agent will bind to the relatively less important
       structure containing. . . in destruction of the trypanosome. For
       example, an incoming antibody, with specificity for Replikin structures,
       might attach to the Replikin decoy structure, leaving the true
       Replikin structure remains untouched.
DETD   . . . the glioma Replikin.
TABLE 9
```

Proteins overproduced in scleroderma and associated Replikins:

PMC1 HUMAN:
hreictiqssggimllkdqvlrcskiagvkvaeitelilk (SEQ ID NO. 523)
hreictiqssggimllkdqvlresk (SEQ ID NO. 524)
34KD **nucleolar** scleroderma antigen:
hreictiqssggimllkdqvlrcskiagvkvaeiteliklkalendqk (SEQ ID NO. 525)
hreictiqssggimllkdqvlrcskiagvkvaeitelilk (SEQ ID NO. 526)
Fibrillarin:
kkmqqenmkqpeqltlepyerdh (SEQ ID NO. 527)
kmqqenmkpqeqltlepyerdh (SEQ ID NO. 528)
SPOP HUMAN:
hemeeskknrveindvepevfkemmcfiytgkapnldk (SEQ. . . ID NO. 562)
HP1Hs-alpha protein:
haypedaenkeketak (SEQ ID NO. 563)
keanvkcpqiviafyeerltwh (SEQ ID NO. 564)
kvldrrvvkgqveyllkwkgfseeh (SEQ ID NO. 565)
kgqveyllkwkgfseeh (SEQ ID NO. 566)
FM/Scl **nucleolar** protein:
ksevaagvkksglpsaerlenvlfgphdcsh (SEQ ID NO. 567)
ksevaagvkksgplpsaerlenvlfgph (SEQ ID NO. 568)
kaaeygkkaksetfrllhakniirpqlk (SEQ ID NO. 569)
kaaeygkkaksetfrllhak (SEQ ID NO. 570)
ksetfrllhak (SEQ ID NO.. . .
DETD . . . proteins is another aspect of this ability of other epitopes to interfere with binding of effective anti-Replikin antibodies, since the **decoy** epitopes have many lysine residues, but no histidine residues. Thus, **decoy** epitopes may bind anti-Replikin antibodies, but may keep the antibodies away from histidine-bound respiratory enzymes. Treatment may therefore be most. . .
CLM What is claimed is:
130. The peptide of claim 128 wherein said structural replication-associated peptide is a **nucleolar** scleroderma antigen.

138. The peptide of claim 128 wherein said structural replication-associated peptide is a PM/Scl **nucleolar** peptide.

L11 ANSWER 2 OF 12 USPATFULL on STN
SUMM . . . al. (1993 Proc Natl Acad Sci USA 90, 8000, also incorporated by reference) who combined multiple copies of the HIV **TAR** with an antisense sequence to HIV gag on the same transcript.
SUMM . . . on targets present in a different cellular locale. This was the approach reported by Lisziewicz et al. (1993) where multiple **TAR** sequences, which act to bind the HIV tat protein in the cytoplasm, were present on the same transcript with antisense. . .
SUMM . . . transported to the cytoplasm where it is translated. Other subcellular compartments for localized function include the Golgi apparatus, endoplasmic reticulum, **nucleolus**, mitochondria, chloroplast and the cellular membrane. Thus, a variety of mechanisms exist either to retain macromolecules in specific cellular compartments. . .
DETD . . . binding sequences in the cases where the monomeric units are derived from sequence specific binding proteins such as the HIV **TAR** protein. However, the choice of the sequence of the nucleic acid polymer can be completely unrestricted in cases where the. . .
DETD . . . nucleic acid sequence, as well as combinations of any of these. The protein binding nucleic acid sequence preferably comprises a **decoy** that binds a protein required for viral assembly or viral replication.
DETD . . . a protein binding nucleic acid sequence or combinations of these. Preferred as a protein binding nucleic acid sequence is a **decoy** that binds a protein required for viral assembly or viral

replication.

DETD . . . and a protein binding nucleic acid sequence. As described elsewhere, such a protein binding nucleic acid sequence preferably comprises a **decoy** that binds a protein involved or required for viral assembly or replication. In another aspect of the present composition, the. . .

DETD . . . products can be capable of acting as antisense. The viral or cellular protein can comprise a localizing protein or a **decoy** protein which are described elsewhere. Such localizing proteins preferably comprise a nuclear localizing protein or a cytoplasmic localizing protein. Specific. . .

DETD . . . RNA signals for the dislocation of proteins essential for virus replication. The HIV Rev protein is found principally in the **nucleolus**. However, in the presence of RNA containing RRE sequences, the Rev protein is found principally in the cytoplasm. Therefore, the. . .

DETD [0495] The A segment (from the **tar** sequence of HIV) of target DNA was isolated as described above. This segment was cloned into the Kpn1 BamH1 site. . .

CLM What is claimed is:
133. The composition of claim 132, wherein said protein binding nucleic acid sequence comprises a **decoy** that binds a protein required for viral assembly or viral replication.

153. The composition of claim 152, wherein said protein binding nucleic acid sequence comprises a **decoy** that binds a protein required for viral assembly or viral replication.

170. The composition of claim 169, wherein said protein binding nucleic acid sequence comprises a **decoy** that binds a protein required for a viral assembly or viral replication.

. . . 205. The nucleic acid component of claim 204, wherein said viral or cellular protein comprises a localizing protein or a **decoy** protein.

207. The nucleic acid component of claim 205, wherein said **decoy** protein binds a protein required for viral assembly or viral replication.

L11 ANSWER 3 OF 12 USPATFULL on STN

SUMM . . . al. (1993 Proc Natl Acad Sci USA 90, 8000, also incorporated by reference) who combined multiple copies of the HIV **TAR** with an antisense sequence to HIV gag on the same transcript.

SUMM . . . on targets present in a different cellular locale. This was the approach reported by Lisziewicz et al. (1993) where multiple **TAR** sequences, which act to bind the HIV tat protein in the cytoplasm, were present on the same transcript with antisense. . .

SUMM . . . transported to the cytoplasm where it is translated. Other subcellular compartments for localized function include the Golgi apparatus, endoplasmic reticulum, **nucleolus**, mitochondria, chloroplast and the cellular membrane. Thus, a variety of mechanisms exist either to retain macromolecules in specific cellular compartments. . .

DETD . . . binding sequences in the cases where the monomeric units are derived from sequence specific binding proteins such as the HIV **TAR** protein. However, the choice of the sequence of the nucleic acid polymer can be completely unrestricted in cases where the. . .

DETD . . . nucleic acid sequence, as well as combinations of any of these. The protein binding nucleic acid sequence preferably comprises a **decoy** that binds a protein required for viral assembly or viral replication.

DETD . . . a protein binding nucleic acid sequence or combinations of these. Preferred as a protein binding nucleic acid sequence is a

decoy that binds a protein required for viral assembly or viral replication.

DETD . . . and a protein binding nucleic acid sequence. As described elsewhere, such a protein binding nucleic acid sequence preferably comprises a **decoy** that binds a protein involved or required for viral assembly or replication. In another aspect of the present composition, the. . .

DETD . . . products can be capable of acting as antisense. The viral or cellular protein can comprise a localizing protein or a **decoy** protein which are described elsewhere. Such localizing proteins preferably comprise a nuclear localizing protein or a cytoplasmic localizing protein. Specific. . .

DETD . . . RNA signals for the dislocation of proteins essential for virus replication. The HIV Rev protein is found principally in the **nucleolus**. However, in the presence of RNA containing RRE sequences, the Rev protein is found principally in the cytoplasm. Therefore, the. . .

DETD [0494] The A segment (from the **tar** sequence of HIV) of target DNA was isolated as described above. This segment was cloned into the Kpn1 BamH1 site. . .

CLM What is claimed is:
133. The composition of claim 132, wherein said protein binding nucleic acid seqence comprises a **decoy** that binds a protein required for viral assembly or viral replication.

153. The composition of claim 152, wherein said protein binding nucleic acid sequence comprises a **decoy** that binds a protein required for viral assembly or viral replication.

170. The composition of claim 169, wherein said protein binding nucleic acid sequence comprises a **decoy** that binds a protein required for a viral assembly or viral replication.

. . . 205. The nucleic acid component of claim 204, wherein said viral or cellular protein comprises a localizing protein or a **decoy** protein.

207. The nucleic acid component of claim 205, wherein said **decoy** protein binds a protein required for viral assembly or viral replication.

L11 ANSWER 4 OF 12 USPATFULL on STN

SUMM . . . mutants) can inhibit certain stages of the viral life cycle. A number of anti-HIV suppressors have been reported, such as **decoy** RNA of **TAR** or RRE (Sullenger et al., 1990, Cell 63:601-08; Sullenger et al., 1991, J. Virol. 65:6811-16; Lisziewicz et al., 1993, New. . .

SUMM . . . 1A, TRAP-beta, TID1, HIP, PABP, Cytokine effector-inflammatory response, Nuclear U4A RNA, HnRNP A2/B1, IL-1 beta, TNF-.alpha. receptor, HYPK mRNA, HIV-1 **TAR** binding protein, TRAP-delta, ATP6E, MO25, CD69, Mitochondrial cytochrome oxidase I, Csa-19, 14-3-3 zeta protein, Nip 7-1, EF-1 delta, E16 mRNA,. . .

DETD . . . 1392-1465

protein)			
Keratin related protein (IFN-.gamma. regulated)	AS	X62571	1088-1228
GLUCOSYLTRANSFERASE	S	AJ224875	853-946
Rox (transcriptional repressor)	AS	X96401	4621-4717
p18 protein	S	J04991	556-645
E1c (small **nucleolar** RNA)	S	U12211	17-123
Ferritin heavy subunit	AS	M12937	290-385
p40 (7-transmembrane protein)	AS	Y11395	1168-1286

Accession

H1C SELECTION No. Coordinates

MIP-1.alpha. AS. . . RNA AS V00592
29-88
hnRNP A2/B1 AS D28877 2442-2477
IL-1 beta AS K027701 673-830
TNF-cat receptor AS S63368 2346-2399
HYPK mRNA S AF049613 292-364
HIV-1 **TAR** binding protein S L22453 296-355
TRAP-delta AS Z69043 236-281
ATP6E AS NM001696 1-37
M025 AS AF113536 163-264
CD69 S Z22576 23-141
Mitochondrial cytochrome AS M12548. . .
DETD . . . U4A RNA AS V00592 29-88
hnRNP A2/B1 AS D28877 2442-2477
IL-1 beta AS K027701 673-830
TNF-.alpha.receptor AS S63368 2346-2399
HYPK mRNA S AF049613 292-364
HIV-1 **TAR** binding protein S L22453 296-355
TRAP-delta AS Z69043 236-281
ATP6E AS NM_001696 1-37
MO25 AS AF113536 163-264
CD69 S Z22576 23-141
Mitochondiral cytochrome oxidase I. . .
CLM What is claimed is:
. . . 1A, TRAP-beta, TID1, HIP, PABP, Cytokine effector-inflammatory response, Nuclear U4A RNA, HnRNP A2/B1, IL-1 beta, TNF-.alpha. receptor, HYPK mRNA, HIV-1 **TAR** binding protein, TRAP-delta, ATP6E, MO25, CD69, Mitochondrial cytochrome oxidase I, Csa-19, 14-3-3 zeta protein, Nip 7-1, EF-1 delta, E16 mRNA,. . .

L11 ANSWER 5 OF 12 MEDLINE on STN DUPLICATE 1
AB . . . the cell nucleus thereby allowing transduction of nondividing cells. Using HIV-based lentiviral vectors, we delivered an anti-CCR5 ribozyme (CCR5RZ), a **nucleolar** localizing **TAR** RNA **decoy**, or Pol III-expressed siRNA genes into cultured and primary cells. The CCR5RZ is driven by the adenoviral VA1 Pol III promoter, while the human U6 snRNA Pol III-transcribed **TAR decoy** is embedded in a U16 snoRNA (designated U16TAR), and the siRNAs were expressed from the human U6 Pol III promoter.. . . these vectors ranged from 96-98% in 293 cells to 15-20% in primary PBMCs. A combination of the CCR5RZ and U16TAR **decoy** in a single vector backbone gave enhanced protection against HIV-1 challenge in a selective survival assay in both primary T. . .

L11 ANSWER 6 OF 12 USPATFULL on STN
DETD . . . WD- U28413 296-599
REPEAT PROTEIN (CSA PROTEIN)
HSC70-1NTERACTING PROTEIN U28918
493-737
(PROGESTERONE RECEPTOR-
ASSOCIATED P48 PROTEIN)
T-COMPLEX PROTEIN 1, DELTA U38846
1063-1356
SUBUNIT (TCP- I-DELTA)(CCT-DELTA)
(STIMULATOR OF **TAR** RNA BINDING)
(H5U38846).
7,8-DIHYDRO-8-OXOGUANINE D16581
221-455
TRIPHOSPHATASE (mutT
HOMOMOLOG) (8-OXO-DGTPASE)
(MTH1)
DNA REPAIR PROTEIN XRCC4 U40622
718-969

DNA TOPOISOMERASE III (TOP3) U43431 1534-1785
G/T MISMATCH-SPECIFIC THYMINE U51166
830-1093
DNA. . .
DETD . . . isoform; NOAM Q15829 + P13592;
571 120-kDa isoform (NCAM120) 571824 + X16841
P13593
573 Wilms' tumor protein (WT33; WT1) X51630
P19544
575 120-kDa **nucleolar** protein 1 (NOL1; NOP120) X55504
P46087
576 zinc finger X-chromosomal protein (ZFX) X59738
P17010
thioredoxin peroxidase 2 (TDPX2); thioredoxin-dependent peroxide. . .
DETD . . . multiple tumor
879 suppressor 2 (MTS2) U17075; L36844
P42772
triiodothyronine receptor; thyroid hormone receptor (THRA1); v-erbA-related protein
97 ear-1 M24898
P20393
AF104419 **decoy** receptor 3 (DCR3) AF104419
O95407
B121 dihydrofolate reductase (DHFR) V00507
Q14130; P00374
B122 thymidylate synthase (TYMS; TS) X02308
P04818
octamer-binding transcription factor. . .

L11 ANSWER 7 OF 12 MEDLINE on STN DUPLICATE 2
TI A **nucleolar TAR decoy** inhibitor of HIV-1 replication.
AB . . . in HIV-1 gene expression. It mediates the transactivation of transcription from the HIV-1 LTR by binding to the transactivation response (**TAR**) element in a complex with cyclin T1. Because of its critical and early role in HIV gene expression, Tat and its interaction with the **TAR** element constitute important therapeutic targets for the treatment of HIV-1 infection. Based on the known **nucleolar** localization properties of Tat, we constructed a chimeric small **nucleolar** RNA-**TAR decoy** that localizes to the nucleoli of human cells and colocalizes in the **nucleolus** with a Tat-enhanced GFP fusion protein. When the chimeric RNA was stably expressed in human T lymphoblastoid CEM cells it potently inhibited HIV-1 replication. These results demonstrate that the **nucleolar** trafficking of Tat is critical for HIV-1 replication and suggests a role for the **nucleolus** in HIV-1 viral replication.
CT Check Tags: Human; Support, U.S. Gov't, P.H.S.
*Anti-HIV Agents
Cell Line
Cell Nucleolus: ME, metabolism
Chromosomal Proteins, Non-Histone: ME, metabolism
Gene Expression
Gene Products, tat: GE, genetics
Gene Products, tat: ME,. . . Terminal Repeat: PH, physiology
HIV-1: GE, genetics
*HIV-1: PH, physiology
Intracellular Fluid
Luminescent Proteins: GE, genetics
Luminescent Proteins: ME, metabolism
RNA, Small Nucleolar
Recombinant Fusion Proteins: GE, genetics
Recombinant Fusion Proteins: ME, metabolism
Ribonucleoproteins: ME, metabolism
*Virus Replication

CN 0 (Anti-HIV Agents); 0 (Chromosomal Proteins, Non-Histone); 0 (Gene Products, tat); 0 (Luminescent Proteins); 0 (RNA, Small **Nucleolar**); 0 (Recombinant Fusion Proteins); 0 (Ribonucleoproteins); 0 (fibrillarin)

L11 ANSWER 8 OF 12 USPATFULL on STN

SUMM . . . al. (1993 Proc Natl Acad Sci USA 90, 8000, also incorporated by reference) who combined multiple copies of the HIV **TAR** with an antisense sequence to HIV gag onr the same transcript.

SUMM . . . on targets present in a different cellular locale. This was the approach reported by Lisziewicz et al. (1993) where multiple **TAR** sequences, which act to bind the HIV tat protein in the cytoplasm, were present on the same transcript with antisense. . .

SUMM . . . transported to the cytoplasm where it is translated. Other subcellular compartments for localized function include the Golgi apparatus, endoplasmic reticulum, **nucleolus**, mitochondria, chloroplast and the cellular membrane. Thus, a variety of mechanisms exist either to retain macromolecules in specific cellular compartments. . .

DETD . . . binding sequences in the cases where the monomeric units are derived from sequence specific binding proteins such as the HIV **TAR** protein. However, the choice of the sequence of the nucleic acid polymer can be completely unrestricted in cases where the. . .

DETD . . . nucleic acid sequence, as well as combinations of any of these. The protein binding nucleic acid sequence preferably comprises a **decoy** that binds a protein required for viral assembly or viral replication.

DETD . . . a protein binding nucleic acid sequence or combinations of these. Preferred as a protein binding nucleic acid sequence is a **decoy** that binds a protein required for viral assembly or viral replication.

DETD . . . and a protein binding nucleic acid sequence. As described elsewhere, such a protein binding nucleic acid sequence preferably comprises a **decoy** that binds a protein involved or required for viral assembly or replication. In another aspect of the present composition, the. . .

DETD . . . products can be capable of acting as antisense. The viral or cellular protein can comprise a localizing protein or a **decoy** protein which are described elsewhere. Such localizing proteins preferably comprise a nuclear localizing protein or a cytoplasmic localizing protein. Specific. . .

DETD . . . RNA signals for the dislocation of proteins essential for virus replication. The HIV Rev protein is found principally in the **nucleolus**. However, in the presence of RNA containing RRE sequences, the Rev protein is found principally in the cytoplasm. Therefore, the. . .

DETD [0493] The A segment (from the **tar** sequence of HIV) of target DNA was isolated as described above. This segment was cloned into the Kpn1 BamH1 site. . .

CLM What is claimed is:

133. The composition of claim 132, wherein said protein binding nucleic acid sequence comprises a **decoy** that binds a protein required for viral assembly or viral replication.

153. The composition of claim 152, wherein said protein binding nucleic acid sequence comprises a **decoy** that binds a protein required for viral assembly or viral replication.

170. The composition of claim 169, wherein said protein binding nucleic acid sequence comprises a **decoy** that binds a protein required for a viral assembly or viral replication.

. . . 205. The nucleic acid component of claim 204, wherein said viral or cellular protein comprises a localizing protein or a **decoy** protein.

207. The nucleic acid component of claim 205, wherein said **decoy** protein binds a protein required for viral assembly or viral replication.

L11 ANSWER 9 OF 12 USPATFULL on STN

SUMM . . . al. (1993 Proc Natl Acad Sci USA 90, 8000, also incorporated by reference) who combined multiple copies of the HIV **TAR** with an antisense sequence to HIV gag on: the same transcript.

SUMM . . . on targets present in a different cellular locale. This was the approach reported by Lisziewicz et al. (1993) where multiple **TAR** sequences, which act to bind the HIV tat protein in the cytoplasm, were present on the same transcript with antisense. . .

SUMM . . . transported to the cytoplasm where it is translated. Other subcellular compartments for localized function include the Golgi apparatus, endoplasmic reticulum, **nucleolus**, mitochondria, chloroplast and the cellular membrane. Thus, a variety of mechanisms exist either to retain macromolecules in specific cellular compartments. . .

DETD . . . binding sequences in the cases where the monomeric units are derived from sequence specific binding proteins such as the HIV **TAR** protein. However, the choice of the sequence of the nucleic acid polymer can be completely unrestricted in cases where the. . .

DETD . . . nucleic acid sequence, as well as combinations of any of these. The protein binding nucleic acid sequence preferably comprises a **decoy** that binds a protein required for viral assembly or viral replication.

DETD . . . a protein binding nucleic acid sequence or combinations of these. Preferred as a protein binding nucleic acid sequence is a **decoy** that binds a protein required for viral assembly or viral replication.

DETD . . . and a protein binding nucleic acid sequence. As described elsewhere, such a protein binding nucleic acid sequence preferably comprises a **decoy** that binds a protein involved or required for viral assembly or replication. In another aspect of the present composition, the. . .

DETD . . . products can be capable of acting as antisense. The viral or cellular protein can comprise a localizing protein or a **decoy** protein which are described elsewhere. Such localizing proteins preferably comprise a nuclear localizing protein or a cytoplasmic localizing protein. Specific. . .

DETD . . . RNA signals for the dislocation of proteins essential for virus replication. The HIV Rev protein is found principally in the **nucleolus**. However, in the presence of RNA containing RRE sequences, the Rev protein is found principally in the cytoplasm. Therefore, the. . .

DETD [0490] The A segment (from the **tar** sequence of HIV) of target DNA was isolated as described above. This segment was cloned into the Kpn1 BamH1 site. . .

CLM What is claimed is:

133. The composition of claim 132, wherein said protein binding nucleic acid sequence comprises a **decoy** that binds a protein required for viral assembly or viral replication.

153. The composition of claim 152, wherein said protein binding nucleic acid sequence comprises a **decoy** that binds a protein required for viral assembly or viral replication.

170. The composition of claim 169, wherein said protein binding nucleic acid sequence comprises a **decoy** that binds a protein required for a viral assembly or viral replication.

. . . 205. The nucleic acid component of claim 204, wherein said viral or cellular protein comprises a localizing protein or a **decoy**

protein.

207. The nucleic acid component of claim 205, wherein said **decoy** protein binds a protein required for viral assembly or viral replication.

L11 ANSWER 10 OF 12 USPATFULL on STN

SUMM . . . al. (1993 Proc Natl Acad Sci USA 90, 8000, also incorporated by reference) who combined multiple copies of the HIV **TAR** with an antisense sequence to HIV gag on the same transcript.

SUMM . . . on targets present in a different cellular locale. This was the approach reported by Lisziewicz et al. (1993) where multiple **TAR** sequences, which act to bind the HIV tat protein in the cytoplasm, were present on the same transcript with antisense. . .

SUMM . . . transported to the cytoplasm where it is translated. Other subcellular compartments for localized function include the Golgi apparatus, endoplasmic reticulum, **nucleolus**, mitochondria, chloroplast and the cellular membrane. Thus, a variety of mechanisms exist either to retain macromolecules in specific cellular compartments. . .

DETD . . . binding sequences in the cases where the monomeric units are derived from sequence specific binding proteins such as the HIV **TAR** protein. However, the choice of the sequence of the nucleic acid polymer can be completely unrestricted in cases where the. . .

DETD . . . nucleic acid sequence, as well as combinations of any of these. The protein binding nucleic acid sequence preferably comprises a **decoy** that binds a protein required for viral assembly or viral replication.

DETD . . . a protein binding nucleic acid sequence or combinations of these. Preferred as a protein binding nucleic acid sequence is a **decoy** that binds a protein required for viral assembly or viral replication.

DETD . . . and a protein binding nucleic acid sequence. As described elsewhere, such a protein binding nucleic acid sequence preferably comprises a **decoy** that binds a protein involved or required for viral assembly or replication. In another aspect of the present composition, the. . .

DETD . . . products can be capable of acting as antisense. The viral or cellular protein can comprise a localizing protein or a **decoy** protein which are described elsewhere. Such localizing proteins preferably comprise a nuclear localizing protein or a cytoplasmic localizing protein. Specific. . .

DETD . . . RNA signals for the dislocation of proteins essential for virus replication. The HIV Rev protein is found principally in the **nucleolus**. However, in the presence of RNA containing RRE sequences, the Rev protein is found principally in the cytoplasm. Therefore, the. . .

DETD [0491] The A segment (from the **tar** sequence of HIV) of target DNA was isolated as described above. This segment was cloned into the Kpnl BamH1 site. . .

CLM What is claimed is:

133. The composition of claim 132, wherein said protein binding nucleic acid sequence comprises a **decoy** that binds a protein required for viral assembly or viral replication.

153. The composition of claim 152, wherein said protein binding nucleic acid sequence comprises a **decoy** that binds a protein required for viral assembly or viral replication.

170. The composition of claim 169, wherein said protein binding nucleic acid sequence comprises a **decoy** that binds a protein required for a viral assembly or viral replication.

. . . 205. The nucleic acid component of claim 204, wherein said viral or

cellular protein comprises a localizing protein or a **decoy** protein.

207. The nucleic acid component of claim 205, wherein said **decoy** protein binds a protein required for viral assembly or viral replication.

L11 ANSWER 11 OF 12 USPATFULL on STN

SUMM . . . al. (1993 Proc Natl Acad Sci USA 90, 8000, also incorporated by reference) who combined multiple copies of the HIV **TAR** with an antisense sequence to HIV gag on the same transcript.

SUMM . . . on targets present in a different cellular locale. This was the approach reported by Lisziewicz et al. (1993) where multiple **TAR** sequences, which act to bind the HIV tat protein in the cytoplasm, were present on the same transcript with antisense. . .

SUMM . . . transported to the cytoplasm where it is translated. Other subcellular compartments for localized function include the Golgi apparatus, endoplasmic reticulum, **nucleolus**, mitochondria, chloroplast and the cellular membrane. Thus, a variety of mechanisms exist either to retain macromolecules in specific cellular compartments. . .

DETD . . . binding sequences in the cases where the monomeric units are derived from sequence specific binding proteins such as the HIV **TAR** protein. However, the choice of the sequence of the nucleic acid polymer can be completely unrestricted in cases where the. . .

DETD . . . nucleic acid sequence, as well as combinations of any of these. The protein binding nucleic acid sequence preferably comprises a **decoy** that binds a protein required for viral assembly or viral replication.

DETD . . . a protein binding nucleic acid sequence or combinations of these. Preferred as a protein binding nucleic acid sequence is a **decoy** that binds a protein required for viral assembly or viral replication.

DETD . . . and a protein binding nucleic acid sequence. As described elsewhere, such a protein binding nucleic acid sequence preferably comprises a **decoy** that binds a protein involved or required for viral assembly or replication. In another aspect of the present composition, the. . .

DETD . . . products can be capable of acting as antisense. The viral or cellular protein can comprise a localizing protein or a **decoy** protein which are described elsewhere. Such localizing proteins preferably comprise a nuclear localizing protein or a cytoplasmic localizing protein. Specific. . .

DETD . . . RNA signals for the dislocation of proteins essential for virus replication. The HIV Rev protein is found principally in the **nucleolus**. However, in the presence of RNA containing RRE sequences, the Rev protein is found principally in the cytoplasm. Therefore, the. . .

DETD [0492] The A segment (from the **tar** sequence of HIV) of target DNA was isolated as described above. This segment was cloned into the Kpn1 BamH1 site. . .

CLM What is claimed is:

133. The composition of claim 132, wherein said protein binding nucleic acid sequence comprises a **decoy** that binds a protein required for viral assembly or viral replication.

153. The composition of claim 152, wherein said protein binding nucleic acid sequence comprises a **decoy** that binds a protein required for viral assembly or viral replication.

170. The composition of claim 169, wherein said protein binding nucleic acid sequence comprises a **decoy** that binds a protein required for a viral assembly or viral replication.

. . . 205. The nucleic acid component of claim 204, wherein said viral or cellular protein comprises a localizing protein or a **decoy** protein.

207. The nucleic acid component of claim 205, wherein said **decoy** protein binds a protein required for viral assembly or viral replication.

L11 ANSWER 12 OF 12 USPATFULL on STN

SUMM . . . inhibition by gene therapy, including cellular expression of transdominant mutant gag and env nucleic acids to interfere with virus entry, **TAR** (the binding site for tat, which is typically required for transactivation) decoys to inhibit transcription and trans activation, and RRE. . . . references therein for an overview of HIV infection and the HIV life cycle, gene therapy vectors utilizing ribozymes, antisense molecules, **decoy** genes, transdominant genes and suicide genes, including retroviruses. See also, Yu et al., Gene Therapy (1994) 1:13-26. Antisense and ribozyme. . . .

SUMM . . . it is now discovered that the SL II sequence is bifunctional. As shown herein, the sequence is an effective Rev **decoy**, and, in addition, was shown to be sufficient to direct cellular localization of bifunctional viral inhibitors along the same cellular. . . . the Rev protein multimerizes at a Rev binding site, allowing a single Rev binding nucleic acid to act as a **decoy** for multiple copies of the Rev protein. It is also discovered that the RRE sequence acts as a molecular **decoy**, and can target viral inhibitors to their target viruses. However, quite surprisingly, it is further discovered that the full-length RRE. . . . suitable than RRE subsequences such as the SL II sequence as a viral inhibitor in general, and as a molecular **decoy** in particular.

SUMM . . . the nucleic acid is an RNA, or a nucleic acid which encodes an RNA), which act, inter alia, as molecular **decoy** molecules for Rev.

SUMM . . . is co-localized with the viral nucleic acids. Furthermore, multiple SL II sequences can be used in combination to enhance the **decoy** and targeting effect of the sequences. Thus, in one preferred embodiment, the inhibitor comprises a plurality of SL II sequences. . . .

DRWD . . . is part of a gene therapy vector, which, when expressed, produces an anti-viral RNA which includes an SL II molecular **decoy**. For example, in one preferred embodiment, the inhibitor is a transcription cassette which is encoded by a gene therapy vector. . . . as HIV-1 to replicate in the cell. An inhibitor "encodes" a direct inhibitor such as an active ribozyme, RNA molecular **decoy**, or anti-sense RNA if it contains either the sense or anti-sense coding or complementary nucleic acid which corresponds to the. . . .

DRWD . . . invading virus). In one embodiment, the inhibitor optionally includes nucleic acids which encode separate protein binding sites such as the **TAR** site for Tat binding, and the bound protein.

DRWD . . . a ribozyme would improve efficacy of the ribozyme because such a molecule would be bifunctional (e.g., by providing a nuclease+Rev **decoy** effect). In addition, we hypothesized that ribozyme activity would also be facilitated by linking the ribozyme to the RRE, because. . . .

DRWD The **decoy** effect of the fusion RNA was demonstrated by HIV-1 SF2 infection of a stable cell line, MSLOY-1, expressing the SL. . . . 205:121-126). Therefore, the observed inhibitory effect of the fusion RNA is due to the SL II sequence acting as a **decoy**.

DRWD . . . RRE-containing mRNAs, and distribution of Rev reflects its interaction with RRE-containing RNA and migration of the bound transcript from the **nucleolus** across a solid phase of nucleus and nuclear membrane to the cytoplasm through a specific export pathway (Luznik et al.,. . . .

DRWD . . . take several forms. Typically, the viral inhibitor is a nucleic

acid which has direct anti-viral activity, such as a molecular **decoy**, anti-sense RNA or ribozyme, or indirect anti-viral activity, i.e., where the inhibitor encodes a direct anti-viral activity (e.g., where the. . . sequence). The inhibitors of the invention typically include an SL II nucleic acid, either in its active (i.e., RNA) molecular **decoy** form, or in its encoded form (i.e., in an RNA or DNA vector which encodes the active form). Thus, techniques. .

DRWD Antiviral Agents: antisense nucleic acids, ribozymes, **decoy** nucleic acids and trans-dominant proteins

DRWD . . . Nature, 335:395 (1988). Anti-viral agents which are optionally incorporated into the viral inhibitors of the invention include anti-sense genes, ribozymes, **decoy** genes, and transdominant proteins.

DRWD A **decoy** nucleic acid is a nucleic acid having a sequence recognized by a regulatory nucleic acid binding protein (i.e., a transcription factor, cell trafficking factor, etc.). Upon expression, the transcription factor binds to the **decoy** nucleic acid, rather than to its natural target in the genome. Useful **decoy** nucleic acid sequences include any sequence to which a viral transcription factor binds. For instance, the **TAR** sequence, to which the tat protein binds, and the HIV RRE sequence (in particular the SL II sequence), to which the rev protein binds are suitable sequences to use as **decoy** nucleic acids.

DRWD . . . native form of the protein. For example, tat and rev can be mutated to retain the ability to bind to **TAR** and RRE, respectively, but to lack the proper regulatory function of those proteins. In particular, rev can be made transdominant. . . by the inhibitors of the invention, for instance, in an expression cassette which also includes, e.g., the SL II molecular **decoy** in conjunction with a ribozyme.

DRWD Examples of antisense molecules, ribozymes and **decoy** nucleic acids and their use can be found in Weintraub, Sci. Am., 262:40-46 (January 1990); Marcus-Sekura, Anal. Biochem., 172:289-95 (1988);. . .

DRWD . . . most embodiments of the invention, the active form of the inhibitor is an RNA molecule which acts as a molecular **decoy** for Rev (through binding of Rev to SL II) and as an anti-sense or ribozyme sequence which disrupts normal viral. . .

DRWD . . . inhibitors are placed into expression cassettes which direct expression of the active inhibitors (SL II decoys, ribozymes, anti-sense nucleic acid, **TAR decoy**, transdominant gene and the like). Ideally, expression of the construct should be sufficiently high to inhibit the growth, infection or. . .

DRWD . . . which are necessary for replication of the vector are present in the vector. In HIV, this typically includes, e.g., the **TAR** sequence, the sequences necessary for HIV packaging, the RRE sequence if the instability elements of the p17 gene of gag. . .

DRWD The **TAR** sequence is located in the R portion of the 5' LTR. It is the sequence to which the tat protein. . .

DRWD . . . by trans-complementation which are necessary for replication of the vector are present in the vector. This typically includes, e.g., the **TAR** sequence, the sequences necessary for HIV packaging, the RRE sequence if the instability elements of the p17 gene of gag. . .

DRWD The **TAR** sequence is located in the R portion of the 5' LTR. It is the sequence to which the tat protein. . .

DRWD . . . al. 1) describe anti-sense inhibition of HIV-1 infectivity in target cells using viral vectors with a constitutive expression cassette expressing anti-**TAR** RNA. Chatterjee et al. (PCT application PCT/US91/03440 (1991), hereinafter Chatterjee et al. 2) describe viral vectors, including AAV-based vectors which express antisense **TAR** sequences. Chatterjee and Wong (Methods, A companion to Methods in Enzymology (1993), 5: 51-59) further describe viral vectors for the. .

DRWD . . . inhibitor which includes an SL II nucleic acid, and an anti-viral therapeutic agent (e.g., transdominant gene, ribozyme,

anti-sense gene, and/or **decoy** gene) which inhibits the growth or replication of a virus (e.g., and HIV virus such as HIV-1). The gene therapy. . .

DETD RRE **decoy** effect of the SL II-ribozyme fusion RNA

DETD To specifically examine the RRE **decoy** effect of an SL II-hairpin ribozyme fusion RNA, HIV-1 SF2 was used as a challenge virus for cells expressing anti-U5. . . HIV-1 was not detected in the MSLOY-1 cells. Thus, the protection in the MSLOY-1 cells was due to an RRE **decoy** effect of the fusion molecule. In contrast, HIV-1.sub.SF expression was inhibited in both MMJT and MSLMJT cells, expressing either the. . .

DETD . . . day 31 at 1000:1 infection, or day 25 at 100:1 infection. Thus, a single antiviral gene (ribozyme or SL II **decoy**) had a detectable, inhibitory effect on viral replication. Furthermore, the p24 level was kept at a low level in MSLMJT. . . indicated that the combination of the SL II and ribozyme was more effective than either the ribozyme or SL II **decoy** alone in the inhibition of HIV-1 .

DETD . . . RNA, the reduction in proviral DNA synthesis was measured in the first round of replication after viral challenge. The RRE **decoy** effect is not relevant in this early part of the replication cycle. The proviral DNA level in the stable cell. . . in the proviral DNA copy number was observed between MdMJT and MSLdMJT, suggesting a lack of effect of the RRE **decoy** on preintegration events. The DNA copy number for MSLMJT was reduced to 1/7 of that for MSLdMJT, whereas that for. . .

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Executing the logoff script...

=> LOG H

COST IN U.S. DOLLARS	SINCE FILE ENTRY	TOTAL SESSION
FULL ESTIMATED COST	110.77	110.98

DISCOUNT AMOUNTS (FOR QUALIFYING ACCOUNTS)	SINCE FILE ENTRY	TOTAL SESSION
CA SUBSCRIBER PRICE	-3.91	-3.91

SESSION WILL BE HELD FOR 60 MINUTES
STN INTERNATIONAL SESSION SUSPENDED AT 17:44:08 ON 10 DEC 2003

L11 ANSWER 1 OF 12 USPATFULL on STN
ACCESSION NUMBER: 2003:276389 USPATFULL
TITLE: Replikin peptides and antibodies therefore
INVENTOR(S): Bogoch, Samuel, New York, NY, UNITED STATES
Bogoch, Elenore S., New York, NY, UNITED STATES

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 2003194414	A1	20031016
APPLICATION INFO.:	US 2002-189437	A1	20020708 (10)

RELATED APPLN. INFO.: Continuation-in-part of Ser. No. US 2002-105232, filed on 26 Mar 2002, PENDING Continuation-in-part of Ser. No. US 2001-984057, filed on 26 Oct 2001, PENDING

	NUMBER	DATE
PRIORITY INFORMATION:	US 2001-303396P	20010709 (60)
	US 2001-278761P	20010327 (60)

DOCUMENT TYPE: Utility
FILE SEGMENT: APPLICATION
LEGAL REPRESENTATIVE: KENYON & KENYON, 1500 K STREET, N.W., SUITE 700, WASHINGTON, DC, 20005
NUMBER OF CLAIMS: 173
EXEMPLARY CLAIM: 1
NUMBER OF DRAWINGS: 9 Drawing Page(s)
LINE COUNT: 7266
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB The present invention provides a new class of peptides related to rapid replication and their use in diagnosing, preventing and treating disease.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L11 ANSWER 2 OF 12 USPATFULL on STN
ACCESSION NUMBER: 2003:152954 USPATFULL
TITLE: NON-NATIVE POLYMERASE ENCODING NUCLEIC ACID CONSTRUCT
INVENTOR(S): RABBBANI, ELAZAR, NEW YORK, NY, UNITED STATES
STAVRIANOPOULOS, JANNIS G., BAY SHORE, NY, UNITED STATES
DONEGAN, JAMES J., LONG BEACH, NY, UNITED STATES
LIU, DAKAI, BETHPAGE, NY, UNITED STATES
KELKER, NORMAN E., NEW YORK, NY, UNITED STATES
ENGELHARDT, DEAN L., NEW YORK, NY, UNITED STATES

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 2003104620	A1	20030605
APPLICATION INFO.:	US 1997-978636	A1	19971125 (8)

RELATED APPLN. INFO.: Division of Ser. No. US 1995-574443, filed on 15 Dec 1995, ABANDONED
DOCUMENT TYPE: Utility
FILE SEGMENT: APPLICATION
LEGAL REPRESENTATIVE: ENZO DIAGNOSTICS, INC., C/O ENZO BIOCHEM INC., 527 MADISON AVENUE 9TH FLOOR, NEW YORK, NY, 10022
NUMBER OF CLAIMS: 244
EXEMPLARY CLAIM: 1
NUMBER OF DRAWINGS: 51 Drawing Page(s)
LINE COUNT: 5162
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB The present invention provides an array of compositions useful for effecting and/or exhibiting changes in biological functioning and processing within cells and in biological systems containing such cells. In effect, these compositions combine chemical modifications and/or ligand additions with biological functions. The chemical modifications

and/or ligand additions provide additional characteristics to the compositions without interfering substantially with their biological function. Such additional characteristics include nuclease resistance, targeting specific cells or specific cell receptors localizing to specific sites within cells and augmenting interactions between the compositions and target cells of interest as well as decreasing such interactions when desired. Also provided by the present invention are processes and kits.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L11 ANSWER 3 OF 12 USPATFULL on STN
ACCESSION NUMBER: 2003:127221 USPATFULL
TITLE: PROCESS FOR SELECTIVE EXPRESSION OF NUCLEIC ACID PRODUCTS
INVENTOR(S): RABBANI, ELAZAR, NEW YORK, NY, UNITED STATES
STAVRIANOPOULOS, JANNIS G., BAY SHORE, NY, UNITED STATES
DONEGAN, JAMES J., LONG BEACH, NY, UNITED STATES
LIU, DAKAI, BETHPAGE, NY, UNITED STATES
KELKER, NORMAM E., NEW YORK, NY, UNITED STATES
ENGELHARDT, DEAN L., NEW YORK, NY, UNITED STATES

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 2003087434	A1	20030508
APPLICATION INFO.:	US 1997-978635	A1	19971125 (8)

RELATED APPLN. INFO.: Division of Ser. No. US 1995-574443, filed on 15 Dec 1995, ABANDONED
DOCUMENT TYPE: Utility
FILE SEGMENT: APPLICATION
LEGAL REPRESENTATIVE: ENZO THERAPETICS, C/O ENZO BIOCHEM INC, 527 MADISON AVENUE 9TH FLOOR, NEW YORK, NY, 10022
NUMBER OF CLAIMS: 244
EXEMPLARY CLAIM: 1
NUMBER OF DRAWINGS: 50 Drawing Page(s)
LINE COUNT: 4844
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB The present invention provides an array of compositions useful for effecting and/or exhibiting changes in biological functioning and processing within cells and in biological systems containing such cells. In effect, these compositions combine chemical modifications and/or ligand additions with biological functions. The chemical modifications and/or ligand additions provide additional characteristics to the compositions without interfering substantially with their biological function. Such additional characteristics include nuclease resistance, targeting specific cells or specific cell receptors localizing to specific sites within cells and augmenting interactions between the compositions and target cells of interest as well as decreasing such interactions when desired. Also provided by the present invention are processes and kits.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L11 ANSWER 4 OF 12 USPATFULL on STN
ACCESSION NUMBER: 2003:127061 USPATFULL
TITLE: Compositions and methods for inhibiting human immunodeficiency virus infection by down-regulating human cellular genes
INVENTOR(S): Holzmayer, Tanya A., Mountain View, CA, UNITED STATES
Holzmayer, Andrew, Libertyville, IL, UNITED STATES LR
Dunn, Stephen J., Mountain View, CA, UNITED STATES

	NUMBER	KIND	DATE

PATENT INFORMATION:	US 2003087273	A1	20030508
APPLICATION INFO.:	US 2002-186593	A1	20020701 (10)

	NUMBER	DATE
PRIORITY INFORMATION:	US 2001-302157P	20010629 (60)
	US 2001-313252P	20010817 (60)

DOCUMENT TYPE: Utility
FILE SEGMENT: APPLICATION
LEGAL REPRESENTATIVE: MCDONNELL BOEHNEN HULBERT & BERGHOFF, 300 SOUTH WACKER DRIVE, SUITE 3200, CHICAGO, IL, 60606
NUMBER OF CLAIMS: 21
EXEMPLARY CLAIM: 1
LINE COUNT: 1819
CAS INDEXING IS AVAILABLE FOR THIS PATENT.

AB The invention provides methods for identifying human cellular genes and their encoded products for use as targets in the design of therapeutic agents for inhibiting or suppressing human immunodeficiency virus (HIV) infection. The invention also provides methods for identifying protective compounds including immunizing agents that inhibit HIV infection. The invention further provides compounds for use in the treatment or prevention of HIV.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L11 ANSWER 5 OF 12 MEDLINE on STN DUPLICATE 1
ACCESSION NUMBER: 2003372791 IN-PROCESS
DOCUMENT NUMBER: 22789097 PubMed ID: 12907142
TITLE: Inhibition of HIV-1 infection by lentiviral vectors expressing Pol III-promoted anti-HIV RNAs.
AUTHOR: Li Ming-Jie; Bauer Gerhard; Michienzi Alessandro; Yee Jiing-Kuan; Lee Nan-Sook; Kim James; Li Shirley; Castanotto Daniela; Zaia John; Rossi John J
CORPORATE SOURCE: Division of Molecular Biology, Beckman Research Institute of the City of Hope, Duarte, California 91010, USA.
CONTRACT NUMBER: AI29329 (NIAID)
AI42552 (NIAID)
AI46030 (NIAID)
SOURCE: MOLECULAR THERAPY, (2003 Aug) 8 (2) 196-206.
Journal code: 100890581. ISSN: 1525-0016.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: IN-PROCESS; NONINDEXED; Priority Journals
ENTRY DATE: Entered STN: 20030809
Last Updated on STN: 20030819

AB A primary advantage of lentiviral vectors is their ability to pass through the nuclear envelope into the cell nucleus thereby allowing transduction of nondividing cells. Using HIV-based lentiviral vectors, we delivered an anti-CCR5 ribozyme (CCR5RZ), a **nucleolar** localizing **TAR** RNA **decoy**, or Pol III-expressed siRNA genes into cultured and primary cells. The CCR5RZ is driven by the adenoviral VA1 Pol III promoter, while the human U6 snRNA Pol III-transcribed **TAR** **decoy** is embedded in a U16 snoRNA (designated U16TAR), and the siRNAs were expressed from the human U6 Pol III promoter. The transduction efficiencies of these vectors ranged from 96-98% in 293 cells to 15-20% in primary PBMCs. A combination of the CCR5RZ and U16TAR **decoy** in a single vector backbone gave enhanced protection against HIV-1 challenge in a selective survival assay in both primary T cells and CD34(+)-derived monocytes. The lentiviral vector backbone-expressed siRNAs also showed potent inhibition of p24 expression in PBMCs challenged with HIV-1. Overall our results demonstrate that the lentiviral-based vectors can efficiently deliver single constructs as well as combinations

of Pol III therapeutic expression units into primary hematopoietic cells for anti-HIV gene therapy and hold promise for stem or T-cell-based gene therapy for HIV-1 infection.

L11 ANSWER 6 OF 12 USPATFULL on STN
ACCESSION NUMBER: 2002:16850 USPATFULL
TITLE: Human stress array
INVENTOR(S): Chenchik, Alex, Palo Alto, CA, UNITED STATES
Lukashev, Matvey E., Newton, MA, UNITED STATES

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 2002009730	A1	20020124
APPLICATION INFO.:	US 2001-782909	A1	20010213 (9)

RELATED APPLN. INFO.: Continuation-in-part of Ser. No. US 1999-441920, filed on 17 Nov 1999, UNKNOWN
DOCUMENT TYPE: Utility
FILE SEGMENT: APPLICATION
LEGAL REPRESENTATIVE: Bret E. Field, BOZICEVIC, FIELD & FRANCIS LLP, 200 Middlefield Road, Suite 200, Menlo Park, CA, 94025
NUMBER OF CLAIMS: 36
EXEMPLARY CLAIM: 1
LINE COUNT: 2377
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB Human stress arrays and methods for their use are provided. The subject arrays include a plurality of polynucleotide spots, each of which is made up of a polynucleotide probe composition of unique polynucleotides corresponding to a human stress gene. The subject arrays find use in hybridization assays, particularly in assays for the identification of differential gene expression of human stress genes.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L11 ANSWER 7 OF 12 MEDLINE on STN DUPLICATE 2
ACCESSION NUMBER: 2002647867 MEDLINE
DOCUMENT NUMBER: 22294961 PubMed ID: 12376617
TITLE: A **nucleolar TAR decoy** inhibitor of HIV-1 replication.
AUTHOR: Michienzi Alessandro; Li Shirley; Zaia John A; Rossi John J
CORPORATE SOURCE: Divisions of Molecular Biology and Virology, Beckman Research Institute of the City of Hope, 1450 East Duarte Road, Duarte, CA 91010-3011, USA.
CONTRACT NUMBER: AI29329 (NIAID)
AI46030 (NIAID)
SOURCE: PROCEEDINGS OF THE NATIONAL ACADEMY OF SCIENCES OF THE UNITED STATES OF AMERICA, (2002 Oct 29) 99 (22) 14047-52. Journal code: 7505876. ISSN: 0027-8424.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200212
ENTRY DATE: Entered STN: 20021031
Last Updated on STN: 20030105
Entered Medline: 20021209
AB Tat is a critical regulatory factor in HIV-1 gene expression. It mediates the transactivation of transcription from the HIV-1 LTR by binding to the transactivation response (**TAR**) element in a complex with cyclin T1. Because of its critical and early role in HIV gene expression, Tat and its interaction with the **TAR** element constitute important therapeutic targets for the treatment of HIV-1 infection. Based on the known **nucleolar** localization properties of Tat, we constructed a chimeric small **nucleolar** RNA-**TAR decoy** that localizes to the nucleoli of human cells and colocalizes in the

nucleolus with a Tat-enhanced GFP fusion protein. When the chimeric RNA was stably expressed in human T lymphoblastoid CEM cells it potently inhibited HIV-1 replication. These results demonstrate that the **nucleolar** trafficking of Tat is critical for HIV-1 replication and suggests a role for the **nucleolus** in HIV-1 viral replication.

L11 ANSWER 8 OF 12 USPATFULL on STN
ACCESSION NUMBER: 2001:109885 USPATFULL
TITLE: NOVEL PROPERTY EFFECTING AND/OR PROPERTY EXHIBITING COMPOSITIONS FOR THERAPEUTIC AND DIAGNOSTIC USES
INVENTOR(S): RABBANI, ELAZAR, NEW YORK, NY, United States
STAVRIANOPOULOS, JANNIS G., BAY SHORE, NY, United States
DONEGAN, JAMES J., LONG BEACH, NY, United States
LIU, DAKAI, BETHPAGE, NY, United States
KELKER, NORMAN E., NEW YORK, NY, United States
ENGLEHARDT, DEAN L., NEW YORK, NY, United States

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 2001007767	A1	20010712
APPLICATION INFO.:	US 1997-978632	A1	19971125 (8)

RELATED APPLN. INFO.: Continuation of Ser. No. US 1995-574443, filed on 15 Dec 1995, ABANDONED
DOCUMENT TYPE: Utility
FILE SEGMENT: APPLICATION
LEGAL REPRESENTATIVE: RONALD C FEDUS, ENZO BIOCHEMICAL INC., 527 MADISON AVENUE, 9TH FLOOR, NEW YORK, NY, 10022
NUMBER OF CLAIMS: 244
EXEMPLARY CLAIM: 1
NUMBER OF DRAWINGS: 51 Drawing Page(s)
LINE COUNT: 4848
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB The present invention provides an array of compositions useful for effecting and/or exhibiting changes in biological functioning and processing within cells and in biological systems containing such cells. In effect, these compositions combine chemical modifications and/or ligand additions with biological functions. The chemical modifications and/or ligand additions provide additional characteristics to the compositions without interfering substantially with their biological function. Such additional characteristics include nuclease resistance, targeting specific cells or specific cell receptors localizing to specific sites within cells and augmenting interactions between the compositions and target cells of interest as well as decreasing such interactions when desired. Also provided by the present invention are processes and kits.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L11 ANSWER 9 OF 12 USPATFULL on STN
ACCESSION NUMBER: 2001:105197 USPATFULL
TITLE: NOVEL PROPERTY EFFECTING AND/OR PROPERTY EXHIBITING COMPOSITIONS FOR THERAPEUTIC AND DIAGNOSTIC USES
INVENTOR(S): RABBANI, ELAZAR, NEW YORK, NY, United States
STAVRIANOPOULOS, JANNIS G., BAY SHORE, NY, United States
DONEGAN, JAMES J., LONG BEACH, NY, United States
LIU, DAKAI, BETHPAGE, NY, United States
KELKER, NORMAN E., NEW YORK, NY, United States
ENGELHARDT, DEAN L., NEW YORK, NY, United States

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 2001006816	A1	20010705

APPLICATION INFO.: US 1997-978637 A1 19971125 (8)
RELATED APPLN. INFO.: Division of Ser. No. US 1995-574443, filed on 15 Dec 1995, ABANDONED
DOCUMENT TYPE: Utility
FILE SEGMENT: APPLICATION
LEGAL REPRESENTATIVE: RONALD C FEDUS, ENZO DIAGNOSTICS INC, ENZO BIOCHEM INC, 527 MADISON AVENUE 9TH FLOOR, NEW YORK, NY, 10022
NUMBER OF CLAIMS: 244
EXEMPLARY CLAIM: 1
NUMBER OF DRAWINGS: 51 Drawing Page(s)
LINE COUNT: 4831
CAS INDEXING IS AVAILABLE FOR THIS PATENT.

AB The present invention provides an array of compositions useful for effecting and/or exhibiting changes in biological functioning and processing within cells and in biological systems containing such cells. In effect, these compositions combine chemical modifications and/or ligand additions with biological functions. The chemical modifications and/or ligand additions provide additional characteristics to the compositions without interfering substantially with their biological function. Such additional characteristics include nuclease resistance, targeting specific cells or specific cell receptors localizing to specific sites within cells and augmenting interactions between the compositions and target cells of interest as well as decreasing such interactions when desired. Also provided by the present invention are processes and kits.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L11 ANSWER 10 OF 12 USPATFULL on STN
ACCESSION NUMBER: 2001:105196 USPATFULL
TITLE: NOVEL PROPERTY EFFECTING AND/OR PROPERTY EXHIBITING COMPOSITIONS FOR THERAPEUTIC AND DIAGNOSTIC USES
INVENTOR(S): RABBANI, ELAZAR, NEW YORK, NY, United States
STAVRIANOPOULOS, JANNIS G., BAY SHORE, NY, United States
DONEGAN, JAMES J., LONG BEACH, NY, United States
LIU, DAKAI, BETHPAGE, NY, United States
KELKER, NORMAN E., NEW YORK, NY, United States
ENGELHARDT, DEAN L., NEW YORK, NY, United States

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 2001006815	A1	20010705
APPLICATION INFO.:	US 1997-978634	A1	19971125 (8)

RELATED APPLN. INFO.: Continuation of Ser. No. US 1995-574443, filed on 15 Dec 1995, ABANDONED
DOCUMENT TYPE: Utility
FILE SEGMENT: APPLICATION
LEGAL REPRESENTATIVE: RONALD C FEDUS, ENZO DIAGNOSTICS INC, 527 MADISON AVENUE, 9TH FLOOR, NEW YORK, NY, 10022
NUMBER OF CLAIMS: 244
EXEMPLARY CLAIM: 1
NUMBER OF DRAWINGS: 51 Drawing Page(s)
LINE COUNT: 4845
CAS INDEXING IS AVAILABLE FOR THIS PATENT.

AB The present invention provides an array of compositions useful for effecting and/or exhibiting changes in biological functioning and processing within cells and in biological systems containing such cells. In effect, these compositions combine chemical modifications and/or ligand additions with biological functions. The chemical modifications and/or ligand additions provide additional characteristics to the compositions without interfering substantially with their biological function. Such additional characteristics include nuclease resistance, targeting specific cells or specific cell receptors localizing to

specific sites within cells and augmenting interactions between the compositions and target cells of interest as well as decreasing such interactions when desired. Also provided by the present invention are processes and kits.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L11 ANSWER 11 OF 12 USPATFULL on STN
ACCESSION NUMBER: 2001:105195 USPATFULL
TITLE: NOVEL PROPERTY EFFECTING AND/ OR PROPERTY EXHIBITING COMPOSITIONS FOR THERAPEUTIC AND DIAGNOSTIC USES
INVENTOR(S): RABBANI, ELAZAR, NEW YORK, NY, United States
STAVRIANOPOULOS, JANNIS G., BAY SHORE, NY, United States
DONEGAN, JAMES J., LONG BEACH, NY, United States
LIU, DAKAI, BETHPAGE, NY, United States
KELKER, NORMAN E., NEW YORK, NY, United States
ENGELHARDT, DEAN L., NEW YORK, NY, United States

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 2001006814	A1	20010705
APPLICATION INFO.:	US 1997-978633	A1	19971125 (8)

RELATED APPLN. INFO.: Division of Ser. No. US 1995-574443, filed on 15 Dec 1995, ABANDONED
DOCUMENT TYPE: Utility
FILE SEGMENT: APPLICATION
LEGAL REPRESENTATIVE: RONALD C. FEDUS, ENZO DIAGNOSTICS, INC, C/O ENZO BIOCHEM, INC, 527 MADISON AVENUE (9TH FLOOR), NEW YORK, NY, 10022
NUMBER OF CLAIMS: 244
EXEMPLARY CLAIM: 1
NUMBER OF DRAWINGS: 51 Drawing Page(s)
LINE COUNT: 4847
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB The present invention provides an array of compositions useful for effecting and/or exhibiting changes in biological functioning and processing within cells and in biological systems containing such cells. In effect, these compositions combine chemical modifications and/or ligand additions with biological functions. The chemical modifications and/or ligand additions provide additional characteristics to the compositions without interfering substantially with their biological function. Such additional characteristics include nuclease resistance, targeting specific cells or specific cell receptors localizing to specific sites within cells and augmenting interactions between the compositions and target cells of interest as well as decreasing such interactions when desired. Also provided by the present invention are processes and kits.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L11 ANSWER 12 OF 12 USPATFULL on STN
ACCESSION NUMBER: 1999:117339 USPATFULL
TITLE: Chimeric antiviral agents comprising Rev binding nucleic acids and trans-acting ribozymes, and molecules encoding them
INVENTOR(S): Kraus, Gunter, Miami, FL, United States
Wong-Staal, Flossie, San Diego, CA, United States
Yu, Mang, San Diego, CA, United States
Yamada, Osamu, Kobe, Japan
PATENT ASSIGNEE(S): The Regents of the University of California, Oakland, CA, United States (U.S. corporation)

	NUMBER	KIND	DATE

PATENT INFORMATION:	US 5958768	19990928
APPLICATION INFO.:	US 1996-697324	19960823 (8)

	NUMBER	DATE
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CAS INDEXING IS AVAILABLE FOR THIS PATENT.

AB Methods and compositions for the treatment and diagnosis of infections of Rev-binding primate lentiviruses are provided. These methods and compositions utilize the ability of Rev binding nucleic acids such as the SLII sequence from the HIV-1 Rev response element (RRE) to target therapeutic agents to the same sub-cellular location as primate lentiviruses which contain RRE sequences. In particular, the invention provides trans-acting ribozymes comprising Rev-binding nucleic acids less toxic than a full-length RRE, and molecules encoding them. The use of the compositions of the invention as components of diagnostic assays, as prophylactic reagents, and in vectors is also described.